TO STUDY THE NORMATIVE DATAS OF VESTIBULAR EVOKED MYOGENIC POTENTIAL AND COMPARE WITH MIGRAINE PATIENTS

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CHENNAI – 600032

APRIL 2013

CERTIFICATE

This is to certify that the dissertation entitled ***"TO STUDY THE NORMATIVE DATAS OF VESTIBULAR EVOKED MYOGENIC POTENTIAL AND COMPARE WITH MIGRAINE PATIENTS"*** by the candidate **Dr. S. Anbuselvi Mattuvar Kuzhali** for M.D Physiology is a bonafide record of the research done by her during the period of study (2010 – 2013) in the Institute of Physiology and Experimental Medicine, Madras Medical College, Chennai – 600003.

DEAN
Madras Medical College
Chennai-600 003.

DIRECTOR AND PROFESSOR
Institute Of Physiology And
Experimental Medicine
Madras Medical College
Chennai-600 003.

DECLARATION

I, **Dr. S. Anbuselvi Mattuvar Kuzhali,** Solemnly declare that the dissertation titled "***TO STUDY THE NORMATIVE DATAS OF VESTIBULAR EVOKED MYOGENIC POTENTIAL AND TO COMPARE WITH AGE AND SEX MATCHED MIGRAINE PATIENTS"*** was done by me at INSTITUTE OF PHYSIOLOGY AND EXPERIMENTAL MEDICINE, MADRAS MEDICAL COLLEGE, Chennai-3, during 2010-2013 under the guidance of my Director Dr.K.PADMA, M.D.

The Dissertaiton is submitted in partial fulfilment of requirement for the award of M.D.Degree (Branch – V) in Physiology to

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY.

Place:

Date: **DR.S.ANBUSELVI MATTUVAR KUZHALI**

Dr.K.PADMA, M.D
Director
Institute of Physiology And Experimental Medicine
Madras Medical College
Chennai-3.

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LIST OF ABBREVIATIONS

µV/V	Micro Volt , (Micro volt / Volume)
5-HT	5-hydroxytryptamine
AA	Arachidonic Acid
AC	Alternating Current
AL	Amplitude of the left ear.
AR	Amplitude of the right ear
B.C	Before Christ
B/L LT	Binaural left
B/L RT	Binaural Right
BC	Bone conduction
BMI	Body mass index
BPPV	Benign Paroxysmal Positional Vertigo
cGMP	Cyclic Guanosine Monophosphate
CSD	Cortical Spreading Depression
cVEMPS	Cervical Vestibular Evoked Myogenic Potential
DC	Direct current
EMG	Electro mogram
ENG/EOG	Electro Nystagmo Graphy / electro-oculography.
EVAR	Earth-vertical axis rotational testing.
Fz	Forehead

GVS	Galvanic vestibular stimulation.
H+	Hydrogen ions
HORT	Head only rotational testing.
Hz	Hertz
IAD	Inter Amplitude Difference Ratio
ICHS	International Classification of Headache Society
IHS	International Headache Society
K+	Potassium ions
KHz	Kilo Hertz
MO	Migraine with aura
ms	Mili seconds
MUP	Motor unit potential
MV	Migrainous vertigo
NHL	Normal Hearing Level
NO	Nitric oxide
OVAR	Off-vertical axis rotation
SCM	Sterno Cleidomastoid muscle
SPL	Sound Pressure Level
SSN	Superior Sagittal Sinus
STB	Short tone burst

STB – VEMP	Short tone burst Vestibular Evoked Myogenic Potential
SVV/SVH	Subjective visual vertical / subjective visual
TGN	Trigeminal Nucleus Caudalis
U/L	Unilateral stimulation
VAR	VEMP asymmetry ratio
VEMP	Vestibular Evoked Myogenic Potential
VNG/ VOG	Video Nystagmo Graphy / video-oculography..

1. INTRODUCTION

1. INTRODUCTION

" The scientist does not study nature because it is useful; he studies it because he delights in it, and he delights in it because it is beautiful. If nature were not beautiful, it would not be worth knowing, and if nature were not worth knowing, life would not be worth living ".

- ***Julies-henri Poincare (1854-1912).***

This simple thought could apply to the field of science where most of the mysterious matters in this universe are still unravelled and unknown. Understanding the basic structural and functional organisation of any organism is still difficult, thus giving a quest for scientific researchers. The eternal human need for hope of relief, for sympathy, and that something should be done, which is felt by those who are suffering is the most of the aspects of medicine that works in a relative and suspicious fashion as there is no definite cause that can be framed for a disease. No disease suffered by a live man can be known, for every living person has his own peculiarities and always has his own peculiar, personal, novel, complicated disease, unknown to medicine , one such mysterious disease that dates back to the very origins of mankind is , a throbbing pain that is mostle one sided and comes with auras or flashes of light is migraine.

1.1. MIGRAINE

Migraine is one of the most interesting illnesses to study, known since the dawn of time, having accompanied mankind throughout its history. The majority of great medical thinkers have expressed opinions about it, something which does not happen with other illnesses.

[1]Headache was accredited as a supernatural possession by an evil spirit during the archean era and for the Greeks. To exorcise the diabolic spirit , 'trepanation' a method of drilling holes in the brain was one of the home remedies used at that time.

[1]Hippocrates recommended blood letting as a treatment and the Romans used opium in beverages as an analgesic . The concepts on the supernatural origins of illnesses resurfaced during the middle ages.

[1] From the 9th to the 13th century, when the Arab culture of Andalusia emerged, medicine steered towards the path of natural science and durng this period some of the causes of migraine were established.

[1] 'Hemikrania', the etymological root of the word migraine is a greek word meaning 'hemi'- half and 'kranion'-skull, "pain on the one side of the head"[18]

Although once in lifetime everyone would have experienced the discomfort that headache or cephalea can cause, only 16% of the population suffers from the intense pain generated by migraine.

Migraine is defined by the international headache society (IHS) as an intermittent, recurrent unilateral disabling headache associated with nausea sensitivity to sounds and light[2].A useful description of migraine is a benign and recurring syndrome of headache associated with other symptoms of neurologic dysfunction in varying admixtures. As the second most common cause of headache often accompanied by nausea and vomiting it afflicts approximately 15% of women and 6% of men.

[3]As per the world health organisation , 28 million people suffer from migraines. Migraines occur about three times more frequently in women than in men. 25% of all women with migraine suffer four or more attacks per month; 35% of migraineurs experience one to four severe attacks per month and 40% of migraineurs experience one or less than one severe attack a month. Each episode of headache lasts from four hours to three days. Occasionally, it lasts longer. Migraine most commonly strikes women of child bearing age between ages 20 and 40 with a slightly higher age range for men. Due to its inconsistent nature approximately 50% of the migraineurs go undiagnosed or mismanaged to this day.

1.2. MIGRAINE AND VESTIBULAR DYSFUNCTION:

Patients with migraine frequently have vestibular complaints ranging from vertigo to less specific symptoms of dizziness, unsteadiness and head motion intolerance[4]. Approximately 16% of the adult population are affected by migraine at some time in their lives[5] and the lifetime prevalence of

‘dizziness’ (comprising both vertigo and non-vestibular dizziness) has been found to be 23% in a large population-based survey[6] . Thus, among the general population , about 3–4% would be expected to have both migraine and ‘dizziness’ by pure coincidence. However, the association of vestibular dysfunction as the mechanism for dizziness in migraineurs have been shown in experimental animal studies.

Moreover in medical literature, the role of vestibular system is to sense the head and body movements and the attitude of the body relative to gravity in order to generate proper motor responses to head , neck and trunk muscle to maintain optimum vision with steady balance[7]. Although patients cannot clearly describe vertigo which presents as a vague symptom , a structured interview during clinical examination is necessary. It can be defined as a transient spinning sensation or a sense of swaying and tilting, exacerbated by movement of the head. Vertigo is intermittent , occurs as a single or recurrent episodes and lasts for seconds, minutes, hours or days and it often accompanies nausea and vomiting.

In individual migraineurs the critical question is whether vertigo is related to migraine or not. Migrainous vertigo (MV) actually concur much more often in migraineurs and was recognized as one of the most common cause of episodic vertigo among neurologists and migraine specialists[11]

Although the classification of IHS[8] has not included migrainous vertigo, till date its specific criteria have been proposed and utilized in many clinical

trials[9.10]. Still a residual grey area remains unknown to substantiate that mild vestibular dysfunction may presents with vertigo.

1.3.VESTIBULAR EVOKED MYOGENIC POTENTIAL:

Diagnostic testing of the vestibular system is an essential component of treating patients with balance dysfunction. The clinical application of vestibular spinal cord reflex registration started with the work developed by halmagyi and Colebatch in 1992[12] which laid the foundation for its use as a diagnostic tool in patients with vestibular disorders. Until recently, testing methods were constrained to evaluate the integrity of the horizontal semicircular canal, which is only a portion of the vestibular system. Recent advances in otolaryngology have afforded clinicians the ability to assess otolith function through Vestibular Evoked Myogenic Potential (VEMP) testing. The American academy of neurology proposed a new neuro-otologic method to provide information about the saccule and the inferior vestibular nerve on each side separately[11] through VEMP.

VEMP is a short latency evoked potential is one of the many sound evoked muscle reflexes or “sonomotor responses” that are believed to be generated from acoustic stimulation of the saccule with loud sound[13]. It is a series of electric waves that are generated by the vestibular pathway when the loud sound transmits through the middle ear , the saccule, the inferior vestibular nerve, the vestibulospinal tract projecting to accessory nucleus and

finally terminating in the cervical muscles[14]. Evoked by acoustic, bone or galvanic stimulation, the VEMP is a biphasic potential that represents the response of the otolith organs to loud stimulation. An adequate sound is delivered to one ear while the muscular activity is recorded in the ipsilateral sternocleidomastoid muscle (SCM). It has been shown that the initial peak of the VEMP response represents the compound action potential (CAP) of the vestibular nerve fibres synchronously activated during the acoustic stimulation with loud sound[15].

In the last decade there have been remarkable strides in unravelling the mystery of the clinical utility of VEMPs as a diagnostic procedure in migraine. Presently there is lack of evidence from well-contolled prospective clinical trials on VEMP testing regarding the management and improvement of clinical outcomes. Standardization of VEMP and methodological issues differs in various studies in literature still remains to be clarified.

When the normal pathological cut-off point is unknown in a consistent manner or when the variables that influence the outcome are numerous and their direction is also unknown, it is necessary to standardize the procedure to interpret the results. Our work was designed to standardize the VEMP response among different age groups . We also intended to study how far the different modes of neck torsion and the placement of reference electrodes at different bony prominences affecting the VEMP parameters. It was also designed to compare the VEMP findings in controls with migrainous patients

with and without vertigo, and lastly to test the sensitivity of VEMP in diagnosing MV. This relatively new procedure may supplement conventional testing in different populations and possibly steer towards previously inaccessible information about the vestibular system.

2.REVIEW OF LITERATURE

2. REVIEW OF LITERATURE

2.1.HISTORY OF MIGRAINE:[1,16]

Migraine one of the oldest known medical disorder was recorded as early as 3000B.C. Ancient Egyptians had made note of it sometime during 1200B.C. Hippocrates in 400B.C had described the visual aura that can precede the migraine headache and the relief offered by vomiting. Aretaeus of Cappadocia was credited as the discoverer of migraines in the second century

The word ‘Migraine’ was derived from the term hemikrania used by Galenus of Pergamon. Abulcasis applied a hot iron to the head or insertion of garlic into an incision made in the temple to relieve migraine.

Hot irons, bloodletting and even witchcraft were part of the treatment in the middle ages. Abu Bakr Mohamed related this headache with different events in the lives of women especially during menopause, following delivery and abortion.

"Megrim" recognized as classic migraine was one of the five major types of headache described in 'Bibliotheca Anatomica, Medic, Chirurgica', in 1712. Ergotamine tart to relieve migraine was advocated by Graham and Wolff in 1938. Vascular theory was proposed by Harold Wolff in 1950, later the neurogenic came into being.

2.2. MIGRAINE HEADACHE:

Migraine is a common neurological disorder, which affects up to 6% of men and 18% of women in the general population [19,20]. Anyone can have an occasional migraine attack, if the triggering factors are strong enough, but migraine attacks must be recurrent before one is defined as a migraine patient. Common types of migraine include migraine with typical aura (MA) and migraine without aura (MO) according to International Headache Society criteria (IHS) [21](International Headache, 1988,Headache Classification Subcommittee of the International Headache, 2004).

2.3. CLASSIFICATION OF MIGRAINE:

2.3.1.International Headache Society Classification of Migraine

- 1.1 Migraine without aura
- 1.2 Migraine with aura
    - 1.2.1 Migraine with typical aura
    - 1.2.2 Migraine with prolonged aura
    - 1.2.3 Familial hemiplegic migraine
    - 1.2.4 Basilar migraine
    - 1.2.5 Migraine aura without headache
    - 1.2.6 Migraine with acute onset aura
- 1.3 Opthalmoplegic migraine
- 1.4 Retinal migraine

- 1.5 Childhood periodic syndromes that may be precursors to or associated with migraine
    - 1.5.1 Benign paroxysmal vertigo of childhood
    - 1.5.2 Alternating hemiplegia of childhood
- 1.6 Complications of migraine
    - 1.6.1 Status migrainous
    - 1.6.2 Migrainous infarction
- 1.7 Migrainous disorder not fulfilling above criteria

2.4. CLINICAL CHARACTERISTICS OF MIGRAINE:

2.4.1. Migraine with and without aura:

Unilateral throbbing headache, accompanied by nausea or vomiting, photophobia and phonophobia are characteristics of migraine attacks. Physical exertion typically worsens the symptoms. [17]About 20% of migraineurs have aura symptoms (MA) preceding a headache which usually starts within an hour after the aura has occurred. [18]In MA, a visual aura with fortification spectrum is the most common aura type, with hemisensoric symptoms next in frequency. Speech disturbances and unilateral weakness are less frequent. Migraine attacks usually last from 4 to 72 hours. Vertigo and dizziness are the common symptoms among migraineurs aura[22]. Recently, migrainous vertigo was evaluated to be the most common cause of spontaneous recurrent vertigo [23], but is not presently included in the IHS criteria.

To identify Migrainous vertigo (MV) atleast two vertigo attacks[24] with one of the following migrainous symptoms such as headache, photophobia, phonophobia, visual or other auras. Mostly MV mimicks benign paroxysmal positional vertigo (BPPV). Unlike BPPV, migrainous vertigo starts early in life have shorter-lasting episodes with frequent recurrences associated with atypical positional nystagmus, and migrainous symptoms[25].

[27]Pathophysiological link exists between migrainous vertigo and symptoms of Meniere's disease. Symptoms of migraine like headache, photophobia aura are also experienced in 45% of the patients with menieres disease[26].

2.5. GENETICS AND PATHOPHYSIOLOGY OF MIGRAINE:

Genetics and environmetal factors [28,29] play a part in the etiology of migraine. First-degree relatives of probands with migrainous aura (MA) have a relative risk of developing MA, whereas their spouses have no risk. The relative risk of MO for first-degree relatives is much lower. Gene identification for the common types of migraine has come and with conflicting results.

Migraine genetics are associated with studies related to dopaminergic system [30-33], the serotonergic system [34-36], mitochondria [37], the endothelial system [38], and homocysteine-related genes [39].

Results of linkage studies in common types of migraine.

Chromosome		Population/ number of families	Disease
19p13	May et al., 1995	German/ 28 families	MA
	Nyholt et al., 1998	Australian/ 4 families	
	Jones et al., 2001	US/ 16 families	
	Terwindt et al., 2001	Dutch/ 36 families	
No linkage to 19p13	Hovatta et al., 1994	Finnish/ 4 families	
	Noble-Topham et al., 2002	Canadian/ 64 families	
	Brugnoni et al., 2002	Italian/ 12 families	
	Wieser et al., 2003	German/ 143 unrelated patients	
	Kaunisto et al., 2004	Finnish/72 families	
1q31	Lea et al., 2002	Australian/ one family	MA and MO
4q24	Wessman et al., 2002	Finnish/ 50 families	MA
4q21	Björnsson et al., 2003	Icelandic/ 103 families	MO
6p12.2-p21.1	Carlsson et al., 2002	Swedish/ one family	MO and MA
11q24	Cader et al., 2003	Canadian/ 43 families	MA
14q21.2-q22.3	Soragna et al., 2003	Italian/ one family	MO
Xq24-q28	Nyholt et al., 2000	Australian/ 2 families	MA and MO

MA, migraine with aura; MO, migraine without aura

2.5.1.PATHOGENESIS OF MIGRAINE:

Although not much is known about why and how the attack actually begins[40-42]more literature are available about the pathogenesis during the attack. . According to the current view headache is preceded by suppression of brain activation and a depolarization wave that propagates across the occipital cortex at rate of 2 to 3-mm/min, this so called migraine is caused by "cortical spreading depression" (CSD) [43]is followed by a failure of brain-ion homeostasis, efflux of excitatory amino acids like K+ from nerve cells, enhanced energy metabolism[44], and transient increases in cortical blood flow followed by sustained flow decreases[45]. Activation of the trigeminovascular system is implicated as the prime pathophysiology of migraine.(Figure 1).

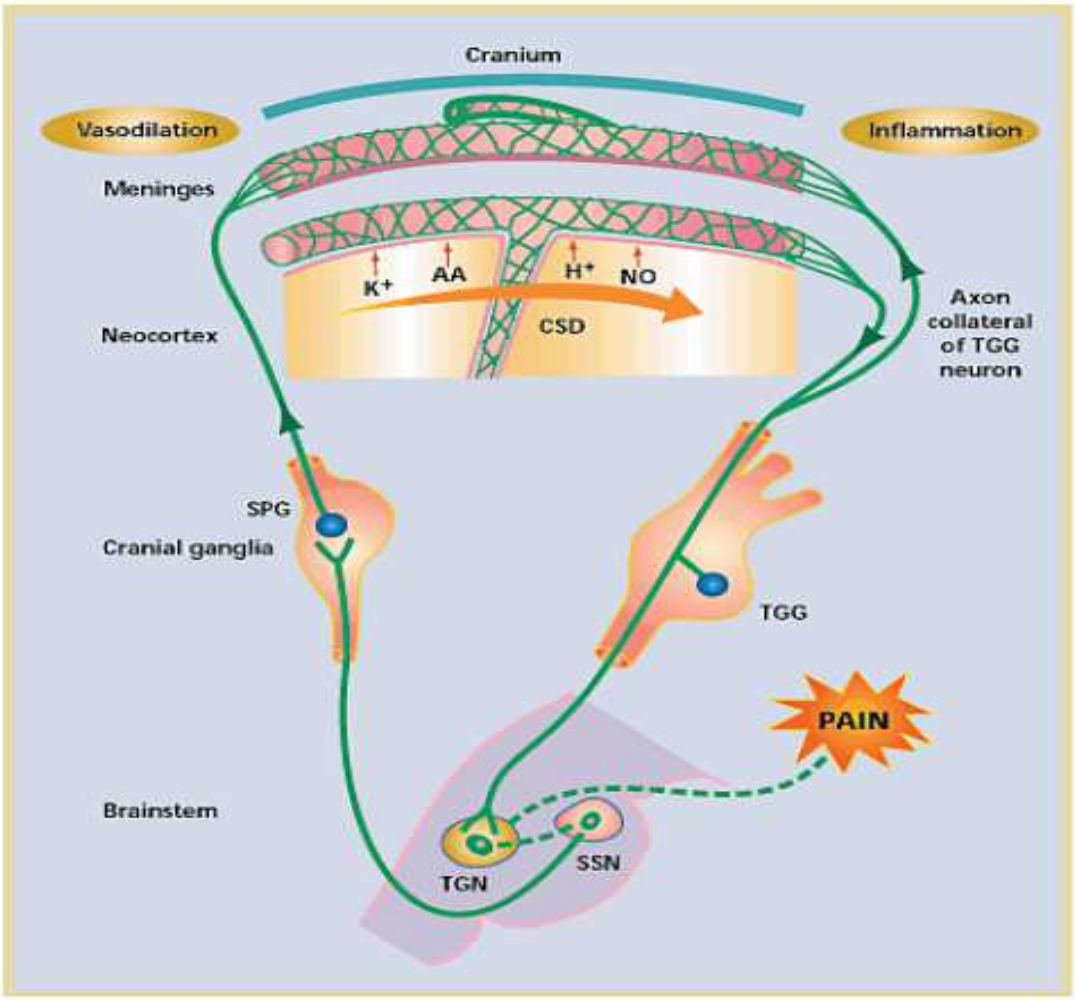
[48]Trigeminal nucleus caudalis via its fibers causes perivascular release of vasoactive neuropeptides which leads to vasodilatation, neurogenic

inflammation causing central transmission of pain impulses leading to headache. A possible link between CSD and trigeminovascular activation in migraine with aura is studied by Bolay et al. In animal models, increased cortical concentration of cyclic guanosine monophosphate (cGMP) and increased extracellular K+ [46] potentiated CSD which inturn induced excess release of nitric oxide (NO) a potent endogenous vasodilatator thus providing an evidence for probable mechanism for recurrence of migraine[47].

Infarction of posterior cerebral territory is commonle seen in migraine mostly affecting the cerebellum[54,55]. Stroke caused by migraine is more common in women[49] and migraine was considered to be an independent risk factor for stroke in women of childbearing age [50-52] especially those with MA [53].

[56] It has been found that Iron homeostasis is progressively impaired in the periaqueductal gray matter of patients with migraine with and without aura. As the periaqueductal gray matter is at the center of the descending antinociceptive neuronal network it is believed to present a possible role in migraine attack generation, potentially by the dysfunction of the trigeminovascular nociceptive system [57].

Figure 1. pathogenesis of migraine :



TGG = trigeminal ganglion; SPG = sphenopalatine ganglion;

SSN = superior sagittal sinus

2.5.2. ROLE OF NEUROMODULATORS:

A.) Serotonin / 5-Hydroxytryptamine:

[58]Reduced systemic 5-HT levels during interictal period and raised levels are seen during migraine attacks. In an animal model serotonin affected the synaptic transmission in the axon terminals of peripheral trigeminovascular neurons and in the cell bodies of central trigeminovascular neurons [58]. The serotoninergic fibres have projections to vestibular nuclei portraying the probable mechanism of vertigo associated with migraine.

B.) Calcium Gated Channels [59]:

. In rats, P/Q-, N-, and L-type voltage-gated calcium channels showed involvement in neurogenic trigeminovascular dural vasodilatation, which may imply involvement in trigeminovascular nociception[60]. P type neuronal calcium channels mediate serotonin release which may cause migraine attack.

C.) Glyceryl Trinitrate:

The initial headache is thought to originate from direct action of the NO-cGMP pathway , while the delayed migraine is likely to result from trigeminovascular activation [62]. Scientific evidence suggests that NO plays an important role in primary headaches [61,63]. It has multiple physiological actions such as endothelium-dependent vasodilatation and neurogenic vasodilatation both of which may be mediated via perivascular nerves [64], and may release relevant neurotransmitters such as calcitonin gene-related peptide (CGRP)

from trigeminal fibers [65-66]. Moreover, NO as a neurotransmitter in the central nervous system (CNS), is of importance in pain perception [67].

D.) Endothelin 1:

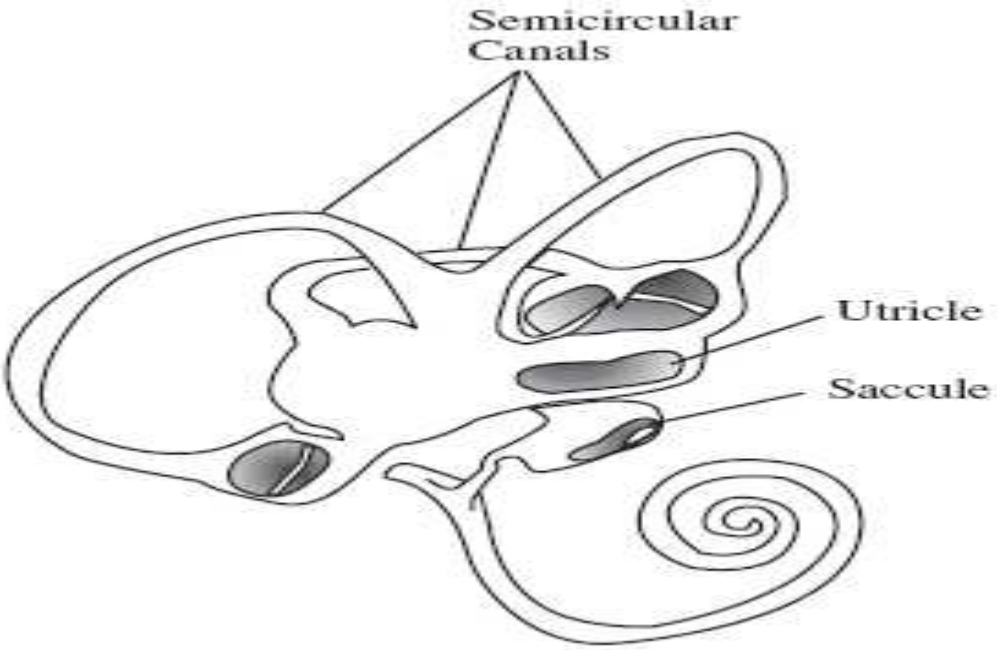
In migraine patients, increased plasma levels of endothelin 1, a potent vasoconstictive peptide, has been reported during ictally [68-70] and interictally. Endothelin 1 stimulation mediate an inhibitory action on NO synthesis by type A receptors in vascular smooth muscle cells [72].

2.6. THE PERIPHERAL AND CENTRAL VESTIBULAR SYSTEM:

[71]The cochlea and the vestibular apparatus are homed in the petrous part of the temporal bone. The organ of corti along with the semicircular canals and otolith organ are integrated in the brain stem and in the cerebellum by means of commissural, inferior olivary, and reverberating circuits.

2.6.1. The Peripheral System[74-75]:

The peripheral vestibular system comprises of the vestibular labyrinth with two otolith organs (the saccule and utricule) and a set of three semicircular canals. These form a coordinate system with the anterior and posterior canal in vertical positions almost orthogonal to each other, while the horizontal canal makes a 30° angle with the horizontal plane.

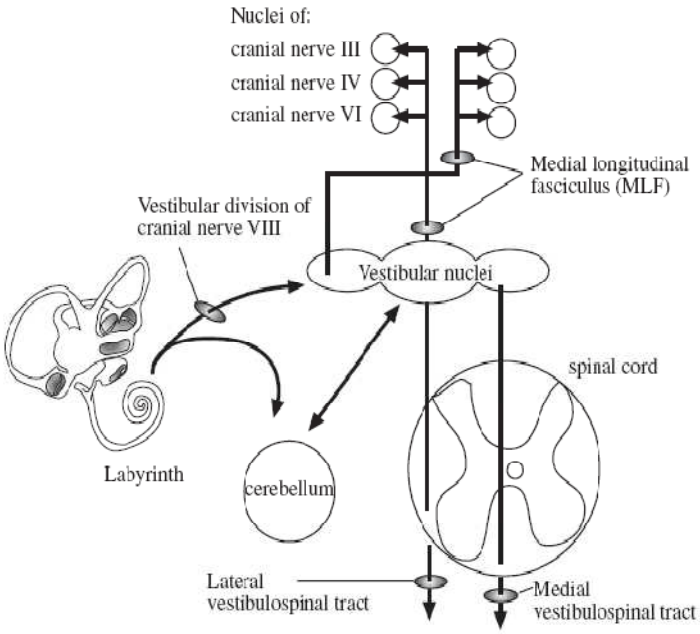


The endolymph movement within the canals stimulates these hair cells and thus transmits a biologic signal to an afferent neuron. The saccular and utricular macules sense linear acceleration, while the cristae of the three semicircular canals sense angular acceleration of the head.

2.6.2. The central vestibular system[76-77]:

The central vestibular system consists of vestibular nuclei in the brain stem, cerebellar-vestibular interaction, vestibulospinal pathways, visual-vestibular interaction, and neck-vestibular interaction. The four vestibular nuclei namely superior, lateral, medial, and inferior are stimulated by afferent fibers from proprioceptive systems and efferent fibres especially from the cerebellum interact with signals from the vestibular organs.

The Central Vestibular System.



Alternate pathways also exist, with chains of interneurons forming reverberating circuits. These interact and fine-tune more specific end-organ reflexes.

2.7. LABORATORY EXAMINATIONS OF THE VESTIBULAR SYSTEM: (is given below).

Eye movement recording	- Electronystagmography / electro-oculography. ENG/ EOG - Videonystagmography / video-oculography VNG/ VOG - Selera search coil
Horizontal semicircular canal	- ocular motor screening - Positional testing - Earth- vertical axis rotational testing. EVAR - Head only rotational testing. HORT
Vertical semicircular canal	- head impulse test - Selera search coil
Utricle	- unilateral centrifugation - off-vertical axis rotation OVAR
Saccule	- VEMP
Vestibular tests with uncertain focus	- Subjective visual vertical / subjective visual horizontal. SVV/SVH - Galvanic vestibular stimulation. GVS

According to Zhou[149] a new possible diagnostic method that are specific for vestibule-spinal pathways and can detect saccular lesions is vestibular evoked myogenic potential.

2.8.VESTIBULAR EVOKED MYOGENIC POTENTIAL:

Vestibular Evoked Myogenic Potential (VEMP) is a short latency biphasic muscle potential that represents the response of the otolith organs when the vestibular system is presented with loud sound. It can also be evoked by acoustic, bone or galvanic stimulation. To produce a reliable VEMP the primary recording site used clinically is the sternocleidomastoid (SCM) along the cervical spine. Recent evolving field in research is VEMP testing as this new procedure may supplement conventional testing in difficult-to-test populations or possibly may be able to evaluate previously inaccessible information about the vestibular system. However one must analyze previous research to fully understand the field, before developing a new clinical test of the vestibular system,

2.8.1. HISTORICAL RESEARCH IN VEMP:-

The sensitivity of the vestibular system to sound was first identified by Pietro Tullio in 1929. Loud sounds when given produced head movements due to vestibular origin that was proposed by von Bekesy [78] in 1935. VEMP evoked by clicks was first recorded from the scalp by [73]Dawson in 1954 followed by Geisler and Rosenblith recorded the same from the occiput in 1962 [74-75]. Shortest latency responses were recorded from the cervical muscles following loud AC tone bursts over the scalp by [76]Bickford in 1963. They were the first to observe that these responses were basically of myogenic in origin

and the amplitude of the response was equally related to the tension in the muscles. [77]A short latency that peaks initially at 13 ms is called as the 'inion response' that are produced by clicks of 120dB and they were foud to be affected by the level of tonic neck extension which then was contradicted by [79] Cody et al. who called it as a 'vertex response' and argued that it was mediated by the cochlea. Although myogenic both the responses differed with frequency and threshold of occurrence[80,83].

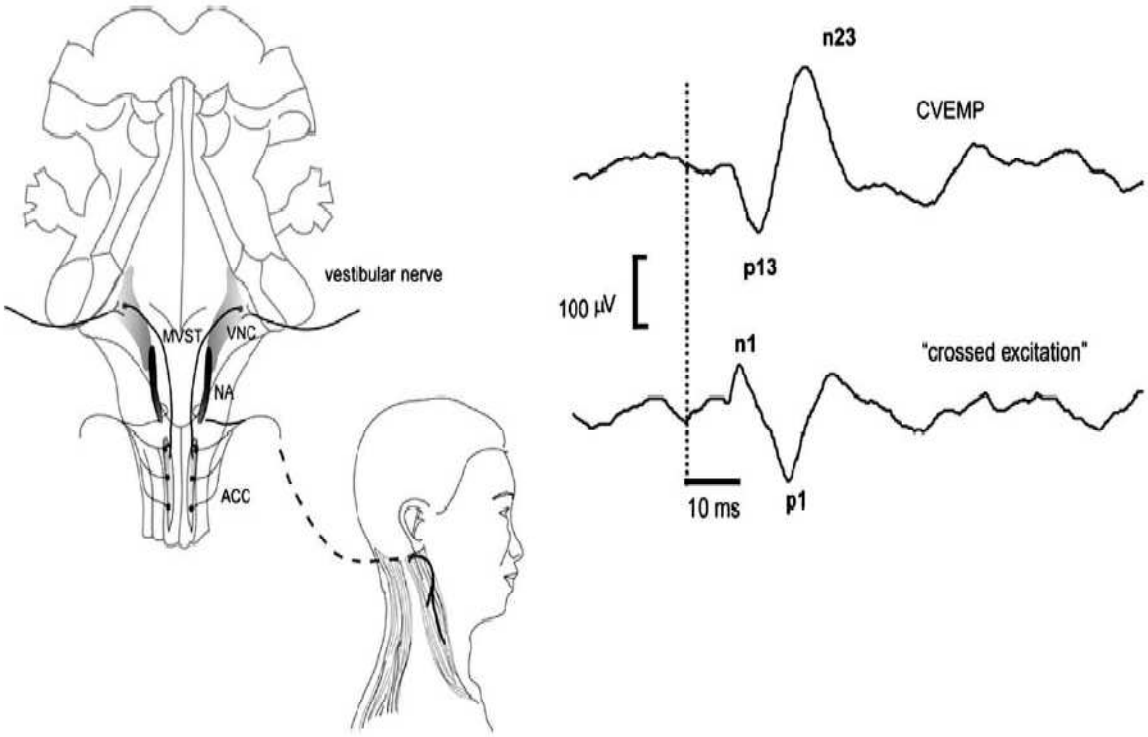
[81-82] The inion response was absent when 'Tack' a procedure which has the tendency to damage the saccule was done by Townsend. As the potential evoked by the background of EMG activity was smaller, it has to be averaged[84] .

[85-86]Colebatch distinguished the short latency response as vestibular-dependent component that arise from the ipsilateral SCM muscle and cochlear-dependent component (crossed response) that arise from the contralateral SCM muscle when loud clicks were given unilaterally. He proposed that the stimulus spread to the utricule that has bilateral projections to the sternocleidomastoid muscle thus producing the crossed response.

Similar bilateral response was also obtained from the tonically active masseter muscles [87]as well as trapezius [88]and splenius capitis [89]. Depending upon the tension developed and the postural control the responses can also be recorded from the triceps and soleus muscles [90-92].

2.8.2. cVEMPs recorded from clicks and tones delivered via headphones :

It is a technique based on testing the residual acoustic sensitivity of the sacculus,



Click evoked VEMP

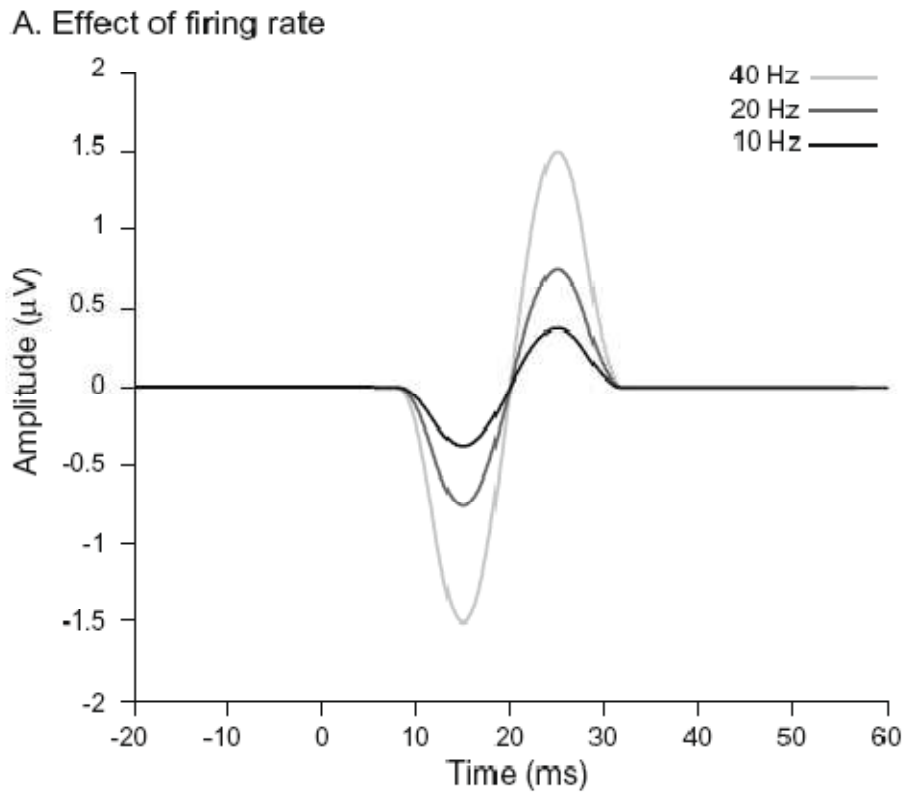
2.8.2.1. Electrogenesis:

An EMG recording of the inhibition of the maximum contracting SCM muscle that represents a short period of multiple motor unit firing averaged together produces the evoked myogenic potential. When the nerve endings of the saccule and utricle are stimulated , the impulses generate inhibitory post synaptic potential in the ipsilateral sternocleidomastoid and the utricle generates excitatory postsynaptic potentials in the contralateral

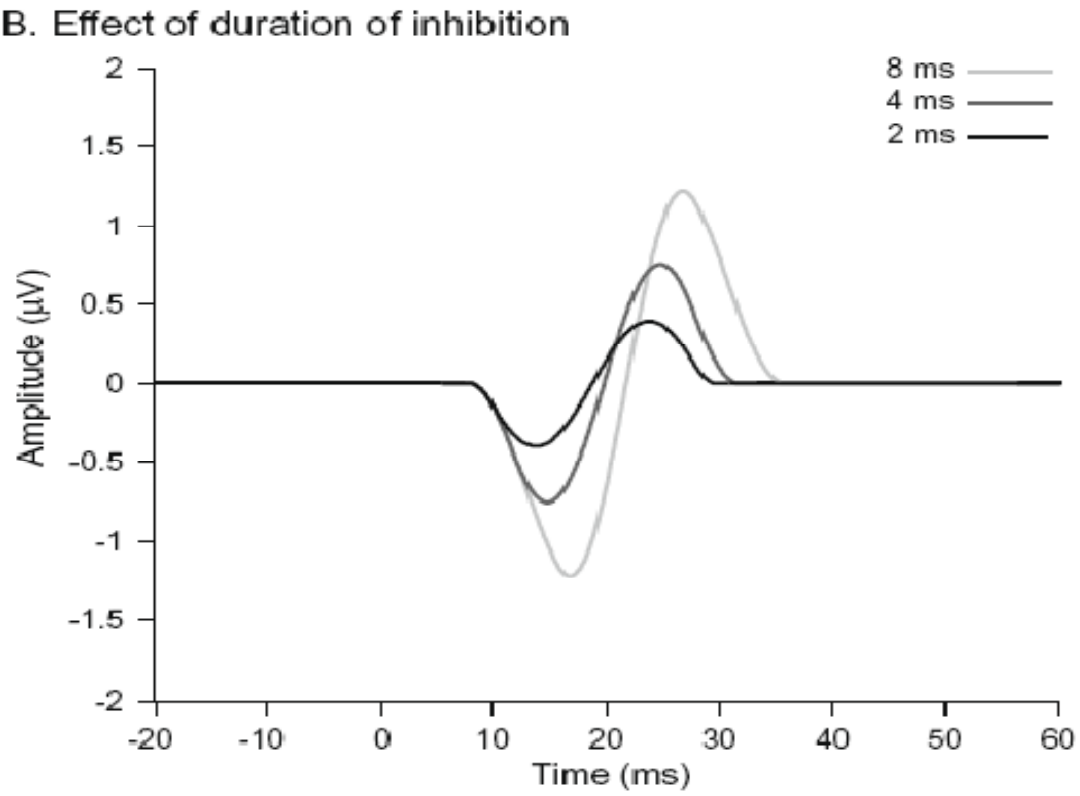
sternocleidomastoid [116]. The time required to inhibit a short period of motor unit firing ranges from 2 to 8ms[118]

A) Effect Of Firing Rate:

[117] A short period of reduced firing probability evoked an inverted motor unit potential (MUP) using unrectified averaging.



[117]When the firing rate was set to be 20ms of muscle contraction (MUP) and a stimulus rate of 5kHz , an amplitude of 20µV (µV peak to peak) was obtained and it directly correlates with the sound intensity and the threshold. When the threshold and intensities are reduced an inverted MUP was obtained explaining that they influence the properties of VEMP.



B) Effect Of Duration Of Inhibition:

With longer durations of the stimulus, there is more duration of inhibition of motor unit and thus prolong the p13 and n23 latencies. The same effect if clinically applied states the reason for the prolonged latencies seen in superior canal dehiscence [119].

2.8.3. Sound stimulus:

A sound intensity of 120 to 140 dB produce eddy current formation in the endolymph of the saccule thereby displaces the hair cells and activates the vestibular afferents[120]. A caliberted source and an intact middle ear to conduct the sound to the end organ is essential for eliciting VEMP successfully. The minimum required stimulus to evoke VEMP was an intense loud clicks or

short tone bursts (STB) of about 95-100 dB above normal hearing level (nHL) that are equivalent to 140-145dB sound pressure level (SPL) and they are considered as the safe limit[85]. For people with above 60 years of age the suggested limit of 95 db nHL and 0.1-millisecond duration are adequate. Hence clicks or short tone bursts are not advisable for patients with tinnitus[85]. With even as small as 10.75dB air-bone gaps VEMP response was not recorded[138]. Threshold levels of the stimulus should be increased as age advances[96].

Clicks are given continuously to each ear at 200 msec intervals (5/second). [122-128] Usually STB and clicks were used to measure VEMP response, though their response rate has not yet been proved clinically.

2.8.4. Amplifier setting[129] :

To evoke a VEMP response without any decrement in the amplitude, the potentials are recorded by averaging 250 presentations with stimulus rates up to 5 Hz/sec, amplified 5000 times and band pass filtered from 30 to 3000 Hz. The optimum frequency was considered between 500 and 1000 Hz. A minimum of two averaged responses are recorded to ensure the reproducibility of VEMP.

2.8.5.BASIC HEAD POSITIONING AND ELECTRODE LAYOUT :

2.8.5.1.PLACEMENT OF ELECTRODES:

As the amplitude of the response depends on the tonic contraction of the muscle, [128]anterior SCM muscle at its middle third was chosen for the active electrode location as it is easily accessibly for recording. Forehead was chosen for the ground electrode and with the reference electrode over the sternum[128].

With subject variations, delayed VEMP responses has been observed for people with fat and long necks as the signal comes from underneath the muscle and from longer distance respectively. Wrists can be used as a reference to avoid volume conduction[120].

2.8.5.2. POSITIONING OF HEAD:

Subjects must lie supine and must activate their SCMs by lifting their heads forwards towards the centre after the sound stimulus was given and the ade- quate levels of tonic neck muscle contraction are maintained during the

recording which corresponding to a mean EMG of about 60μV. Activation of the SCM muscle can also be done by various means like

- pushing the head forward against the resistance of a padded bar or an inflated sphygmomanometer while sitting upright
- lifting head forwards against resistance while lying supine
- turning head away from the source of stimulus while semirecumbent.

[127] for every run of acoustic stimulation subjects are asked to tense the muscle during and relax between runs. To produce less torque for the patient, colebach recommended simply tilting the entire body up by about 30 degrees. Presently very few literatures give amplitude norms for this procedure. The magnitude of the p13-n23 amplitude of VEMP response is largely determined by click intensity and the level of tonic SCM contraction[139] . During recording, if VEMP waveforms are not obtained feedback must be given to subjects to control the levels of neck muscle tension.

2.8.6. VEMP RESPONSE:

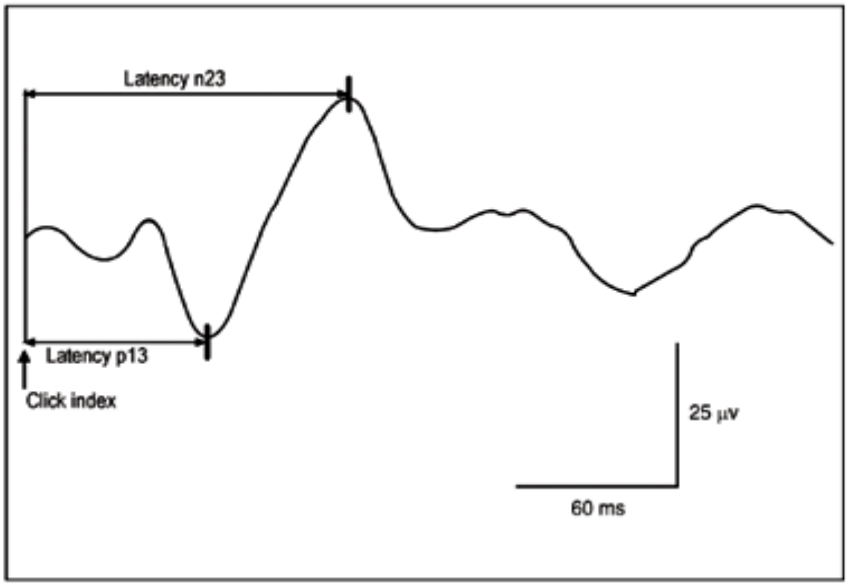
The response evoked in the neck EMG is averaged and presented as a VEMP. The VEMP arises from modulation of background EMG activity and differs from neural potentials in that it requires tonic contraction of the muscle. The latency, amplitude, and threshold for the p13-n23 wave is measured. Though the amplitude is the most reliable measure[121] and latencies are less reliable, latencies are used to confirm a particular waveform as VEMP.

Responses were recordable even in sensorineural hearing loss portraying that it needs the activation of the relevant muscles[130].

[131]The ipsilateral responses are always from the saccule and are inhibitory whereas the contralateral crossed responses are mostly from the utricle and are excitatory. Better ipsilateral and the contralateral responses were seen with click evoked VEMPs and DC or tap evoked VEMPs respectively[132-137].

2.8.6.1. WAVEFORMS OF VEMP:

[135]The response consists of 2 biphasic peaks, p13–n23 and n34–p44 with an initial positivity or inhibition (p13) followed by a negativity or excitation (n23). Later components (n34, p44) have a lower stimulus threshold and are nonvestibular probably cochlear in origin. The short-onset latency of the VEMP (about 8 milliseconds) indicates that it is likely to be mediated by an oligosynaptic pathway, possibly disynaptic and consisting of primary vestibular afferents beginning in the saccule via the inferior vestibular nerve, lateral vestibular nucleus, medial vestibulospinal tract to the accessory nucleus and finally ending at the motor neurons of the SCM muscle[127].



A.) p13 latency[100-103]:

The first negative polarity of the biphasic VEMP waveform is the p13 latency that appears approximately at 13ms. It is prolonged in delayed or incomplete myelination and is significantly longer in newborns than in adults. Due to the structural differences mean p13 latency is shorter in children compated to that of adults.

B.)***n23 latency[104]*** :

The n23 latency is defined as the positive polarity of the biphasic wave that appears approximately at 23ms. The immediate trough following the p13 wave is approximately at 23 ms (n23). Both the latencies of the waveform are affected by the intensity of the sound stimulus and the levels of neck torsion.

C.) p13-n23 amplitude :

[105]The amplitude is defined as the peak-to-peak p13-n23 in µV of the VEMP response and is largely dependent on the click intensity and the level

of tonic SCM contraction.[95] Some authors reported absent response at rest and at 70 years of age. It was found to have a linear correlation with sound intensity above a certain threshold.[127]VEMP amplitude has been considered the reliable parameter as it varies if there is no proper neck torsion or any pathological change that affects the neuro-muscular pathway.

D.) Inter-amplitude difference ratio(IAD) :

[93.94]Also called as VEMP asymmetry ratio (VAR) is defined as the ratio of the sum of the amplitudes of both ears. It is expressed in percentage (%). It signifies the side-to-side differences in reflex amplitude and is expressed as percentage and calculated using the following formula [96]IAD% = (*A*R-*A*L)/(*A*R+ *A*L) * 100, where *A*R and *A*L are amplitude of the right ear and that of the left ear respectively.

E.)Other than p13 and n23 waveforms:

The consecutive wave pattern following p13 and n23 has a trough at the latency near 34 ms (n34) and peaks at about 44 ms (p44) and the waves probably originates from cochlear afferents[133]. Usually, clinical interpretation of a VEMP test includes latency p13, n23, p13-n23 amplitude and interamplitude difference ratio.

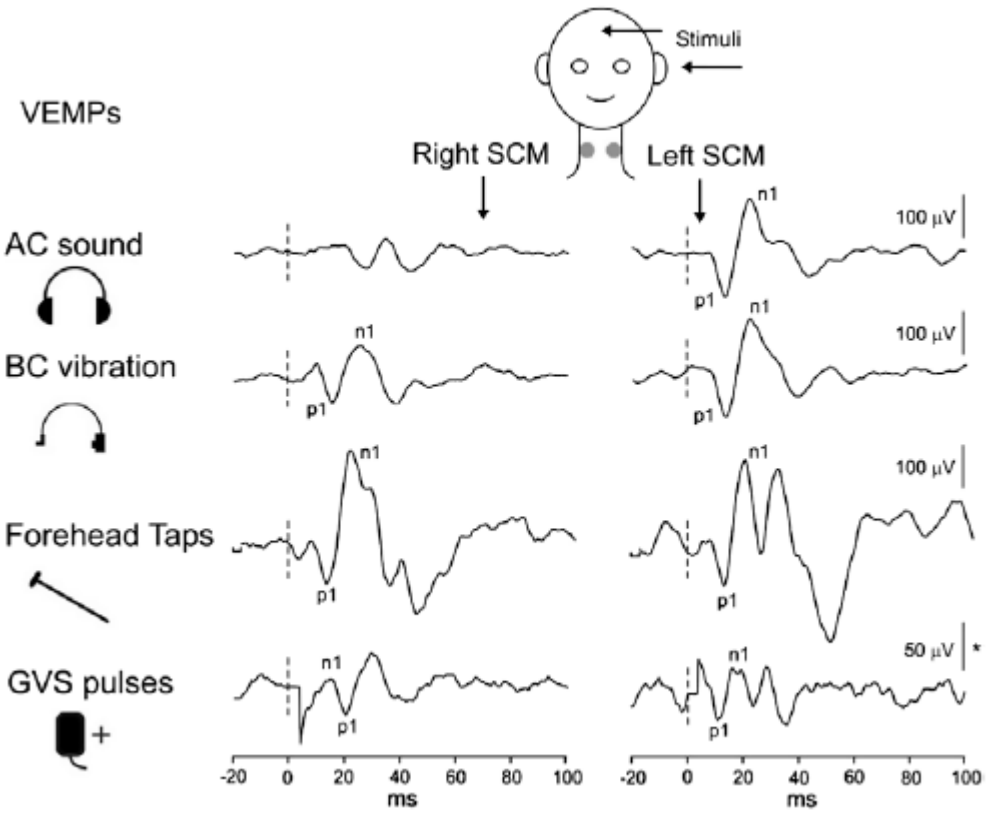
[128-129]The initial positive–negative response (p13n23) is vestibular dependent and strictly ipsilateral to the stimulus. A small contralateral response of opposite polarity, with an initialnegativity (n1p1, crossed neural

response), is present infrequently . These are small, becoming prominent only in the presence of vestibular hypersensitivity to sound.

2.8.7. VEMP RECORDED FROM DIFFERENT STIMULI:

Other stimuli used to evoke cVEMPs are AC sound, skull-taps and BC vibration . cVEMPs decreases in frequency of occurrence and amplitude with age [97-99] inspite of the stimulus type.

[108]AC tone burst produced larger responses due to greater stimulus duration, transmitted sound energy and frequency tuning ompared to click cVEMPs,. The best frequency lies between 200 and 1000 Hz [106-109]. cVEMPs has been elicited by tapping the forehead with a clinical reflex hammer[110]. No variations in the response has been found between the tap evoked and cVEMPs.



[89,107]Using short tone bursts STB VEMPs were recorded from the splenius capitis during tonic contraction of the muscle and their peak latencies found to increase with increasing stimulus duration.

The bone conduction (BC) stimulus reflects the combined otolith projection to the neck [112]. Ipsilateral responses were found to be larger [113] for the BC delivered via the mastoid. Lower frequencies are required to produce BC VEMPs and a larger response can be obtainted at or below 100Hz[111].

Similar contralateral crossed response has been found with cathodal galvanic stimulation GVS[114]. Sometimes an ipsilateral crossed response or an absent response was recorded with anodal stimulation by Watson[115]

2.9. TECHNICAL PITFALLS IN DOING VEMPS:

- Less availability of studies
- Relatively new test
- No protocol and quality assurance.

Hence the concern is to standardize the method according to the population.

A.) assuring neck muscle activation - biggest problem![140]:

Assuring neck muscle activation is a big crisis mainly for two reasons:

- head held up against gravity
- test should be repeated twice to ensure reliability.
- Less comfortable and cant be sustained for longer time.

Although fatiguing only one SCM with the head turned one sided is possible, smaller potentials are produced with less reliable results.

An intrinsic problem arises in case of very young children as it needs continuous co-operation.

B.) Thresholds levels at 500Hz is maintained to produce reliable VEMP[121].

C.) Electrical artefact:

- sound generators near electrodes
- higher impedance
- improper placement of electrodes
- nearby electrical activity

These can produce sinusoidal undulations or stimulus artefact.

D.) Other artifacts:

- Technical errors
- improper instrumentation are to be checked.

2.10.CLINICAL UTILITY OF VEMPs:

VEMP is a promising method for diagnosing and follow up of patients with retrocochlear lesions, brainstem lesions and tumours of vestibular apparatus.

A. IN MENIERES DISEASE[117]:

- The VEMP response is increased in early stage of Menieres disease due to saccular dilatation.
- absent response in advanced cases due to collapse of the saccule.

B. IN RETROCOCHLEAR LESIONS[89,127]:

- More prolonged p13 latency seen in BPPV compared to vestibular neuritis and menieres disease, though their clinical findings were almost the same.
- Asymmetry and long latency response was also found in vestibular neuritis.

C. SPASMODIC TORTICOLLITIS[116]:

- VEMPs are often asymmetrical as reported by Colebatch et al.

D. IN BRAINSTEM LESIONS AND MULTIPLE SCLEROSIS[153]:

- Shimuzu et al reported that both the latencies and the amplitude were prolonged in brainstem stroke , medullary lesions and demyelinating disorders .

3.AIM AND OBJECTIVES

3. AIM :

To assess vestibular evoked myogenic potential in various age groups and compare it with migraine patients.

3.1. OBJECTIVE :

1. To quantify the physiologic changes which occur in the vestibular (specifically saccular pathways) system with aging utilizing the VEMP.
2. To compare vestibular evoked myogenic potentials between two different modes of neck torsion
   (i) lifting head forwards towards centre and
   (ii) lifting head and simultaneously turning away from the ear of stimulus.
3. To evaluate whether the influence of inverting electrode at different sites on the VEMP parameters.
4. To investigate VEMPs in patients with migraine and to compare it with that of age and sex matched controls.

4.MATERIALS AND METHODS

4. MATERIALS AND METHODS:

The study was conducted in the Institute Of Physiology And Experimental Medicine, Madras Medical College, Chennai-3. The study protocol was approved by our Institutional Ethical Committee, Madras Medical College, Chennai. The subjects participating in this study were informed about the study and written consent was obtained from them before including them in the study.

***4.1. STUDY DESIGN*:** It is a cross-sectional study.

Number of groups : Two.

4.2. INCLUSION CRITERIA:

4.2.1. Controls:

Age and sex : 17 years to 70 years of both sex.

Without otologic and neurologic diseases.

4.2.2. Cases:

According to the international headache society ICHS-2 criteria patients who fulfilled the criteria for classical migraine and definite or probable vertiginous migraine were included in this study.

4.3. EXCLUSION CRITERIA:

1. Subjects with conductive deafness either due to ear pathology or any other systemic disorders

2. History of hypertension or diabetes

3. Any neurologic disorder or metabolic disorder.

4.4. SELECTION OF SUBJECTS:

4.4.1. Cases:

50 patients with migraine were selected from the headache outpatient department, Institute of neurology, Madras Medical College, Chennai who fulfilled the ICHS criteria for migraine and definite or probable migrainous vertigo.

4.4.2. Controls:

100 clinically healthy adult volunteers were enrolled as control group in this study. The control group were either the attendants of the migraine patients of RGGH or the students and staff of Madras medical college who attended the study voluntarily.

From the control group,

1. 80 healthy adult volunteers were involved to study the effect of age on VEMP parameters;

2. 25 of the 80 healthy adult volunteers in the age group of 17 to 26 years were involved in the study on the different modes of neck torsion on VEMP parameters.

3. Another 20 healthy adult volunteers of 17 to 26 years were involved to study the influence of reference or inverting electrode placement on VEMP parameters;

4.5. PREPARATION OF SUBJECTS:

STEP 1: Before subjecting to the tests all subjects were examined clinically and the experimental procedure was explained to them in the language that they could understand. An adequate time was given to the subjects to get accustomed to the new settings.

STEP 2: Hearing normality was assessed with otoscopy and pure-tone audiometry

STEP 3: they were then subjected to VEMP test using RMS multichannel polyrite.

The test was done in single session lasting approximately 20 minutes. During the period of testing, participants were given adequate rest periods to reduce fatigue and boredom. The subjects were asked to lie in supine position on the table and the electrodes placed using standard gel (for good conduction) after wiping the area with spirit.

A.) Placement Of Electrodes:

- Ground electrode is placed on the forehead(Fz).
- Reference (inverting) electrode is placed over the sternum.
- Non-inverting or the active electrode is placed over the middle third of the (SCM) sternocleidomastoid muscle.

B.) Stimuli used for recording VEMP:

VEMP recording was initiated when the acoustic stimuli were given as loud air-conducted (AC) sound of short tone bursts 500 HZ via biologic standard RMS headphones (DR-531, RMS MULTIPOLYRITE). The stimulus was presented with rarefaction polarity, two cycles plateau with one cycle rise and fall times. The stimulation rate was 5Hz/sec and the analysis time for each response was 100 ms and 150 responses were averaged for each run. EMG (electromyogenic) activity was recorded both from the ipsilateral and contralateral Ag/AgCl surface electrodes placed over the middle part of the sternocleidomastoid SCM muscle . The EMG activity was amplified 5000 times and band pass filtered 20Hz-2000Hz.

PARAMETERS	**SELECTION**
• STIMULUS	
1. Transducer	Biologic standard RMS headphones
2. Type	500Hz short tone bursts
3. Ramping	Blackman
3.1.Duration	2 cycles plateau; 1 cycle rise/fall
4. Intensity	95-100 dB Nhl
5. Polarity	Rarefaction
6. Rate	5Hz

• ACQUISITION	
1. Analysis time	
1.1. Epoch time	100 ms
2. Electrode type	Surface (Ag/Agcl)
3. Electrode location	
3.1. ground electrode	Forehead
3.2. inverting electrode	Sternum
3.3. non-inverting electrode	Middle third of SCM muscle
4. Band pass filtered	20Hz-2000Hz
5. Amplification	5000 times
6. Averaged for each run	150 presentations.

C.) Procedure of VEMP recording:

Subjects were placed in the supine position. After the auditory click was given subjects were asked to activate their SCMs by lifting their heads forwards towards the centre. They were then asked to maintain adequate levels of tonic neck activation during the recording. Auditory stimulus was given binaurally and monoaurally. The vestibular evoked myogenic potential response was recorded both from the ipsilateral and contralateral SCM muscle both during binaural and monoaural stimulation.

In 20 subjects among the control group , the reference or inverting electrode was also placed at three different sites like sternum, wrist and the mastoid and the VEMP responses were recorded .

In 25 subjects among the control group, VEMP was recorded using two modes of neck torsion like

- Lifting Head Forwards Towards Centre.
- Lifting head and simultaneously turning away from the ear of stimulus

After the recordings were complete, the surface electrodes were removed and the recording paste was wiped with spirit and water.

4.6. *VEMP RESPONSES*

The recorded VEMP showed the following waveforms:

1. The p13 latency - defined as the positive polarity of the biphasic wave that appears at approximately 13 ms

2. The n23 latency - defined as the negative polarity of the biphasic wave that appears at approximately 23 ms.

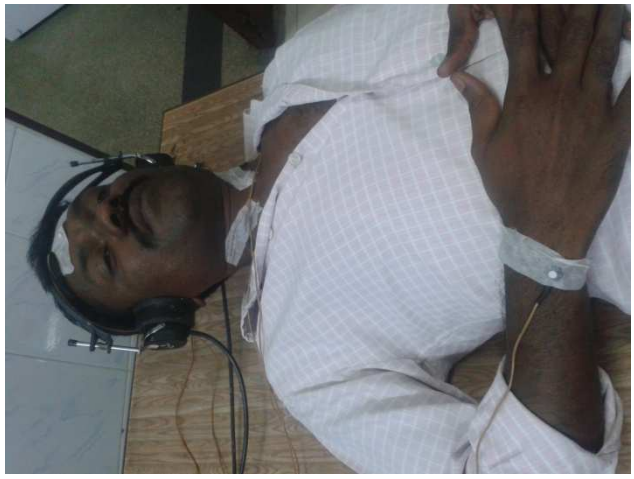
3.The p13-n23 amplitude - defined as the peak-to-peak p13-n23 maximum energy in μV.

4.VEMP asymmetry ratio (VAR) or inter amplitude ratio (IAD) is defined as the ratio of the inter-aural amplitude difference to the sum of the amplitudes of both ears.

RMS MULTICHANNEL POLYRITE

PLACEMENT OF ELECTRODES

STERNUM REFERENCE



WRIST REFERENCE

MASTOID REFERENCE

POSITION OF HEAD

HEAD LIFTED TOWARDS CENTRE

HEAD TURNED AWAY FROM SOURCE OF STIMULUS

5. RESULTS

5. RESULTS

5.1.STATISTICAL ANALYSIS:

The VEMP recordings were conducted on 100 clinically healthy individuals (CONTROLS) and 50 patients with migraine (STUDY GROUP) . The recorded VEMP responses from the control and the migraine study group were statistically analysed and their significance were determined using SPSS for windows under the following headings:

1. Analysis of the anthropometric details between the control and the migraine group by using unpaired students ‘t’ test.
2. Normative data of VEMP in different age groups in the control population by using unpaired students ‘t’ test.
3. Comparison of two modes of neck torsion in the control group by using unpaired students ‘t’ test.
4. Comparison of the different placements of inverting or reference electrode in the control group by One way Analysis Of Variance followed by tukeys multiple comparison tests.
5. Comparison of migraine patients with/without vertigo with age and sex matched controls by using unpaired students ‘t’ test.

5.2.ANALYSIS OF THE ANTHROPOMETRIC DETAILS BETWEEN THE CONTROL AND THE MIGRAINE GROUP.

The anthropometric measures like age, height, weight, BMI of the controls and the migraine subjects were subjected to students unpaired 't' test.

No significant change was found for the controls and the migraine study group and they were found to be suitable for this comparative study. The anthropometric parameters of the control group and the migraine patients were tabulated in table 1.

Table1: Anthropometric measures of the control group and the migraine patients

STUDY GROUP	**AGE Mean ± SD**	**HEIGHT Mean ± SD**	**WEIGHT Mean ± SD**	**BMI Mean ± SD**
Control group N=100	38.9 ± 15.9	162.2 ± 8.03	62 ± 8.8	23.7 ± 3.8
Migraine patients N-50	34.7 ± 11.3	167.2 ± 5.08	68.95 ± 7.6	24.64 ± 2.04
'p' value	0.1 #	0.7 #	0.3 #	0.3 #

 # not significant

5.3. NORMATIVE DATA OF VEMP IN DIFFERENT AGE GROUPS IN THE CONTROL POPULATION:

80 healthy individuals from the control group consisting of 40 males and 40 females in the age group between 17 and 70 years (mean 38.9 ± 15.93) were subjected to VEMP and their results were statistically analysed to derive significance using appropriate students t test. The mean height(cm), weight (kg) and BMI (cm/kg) of the control group was found to be 162.2 ± 8.03, 61.98 ± 8.75, 23.7 ± 3.8 respectively. The p13 latency, n23 latency and p13-n23 amplitude of the VEMP response among different age groups has been tabulated in table 2.

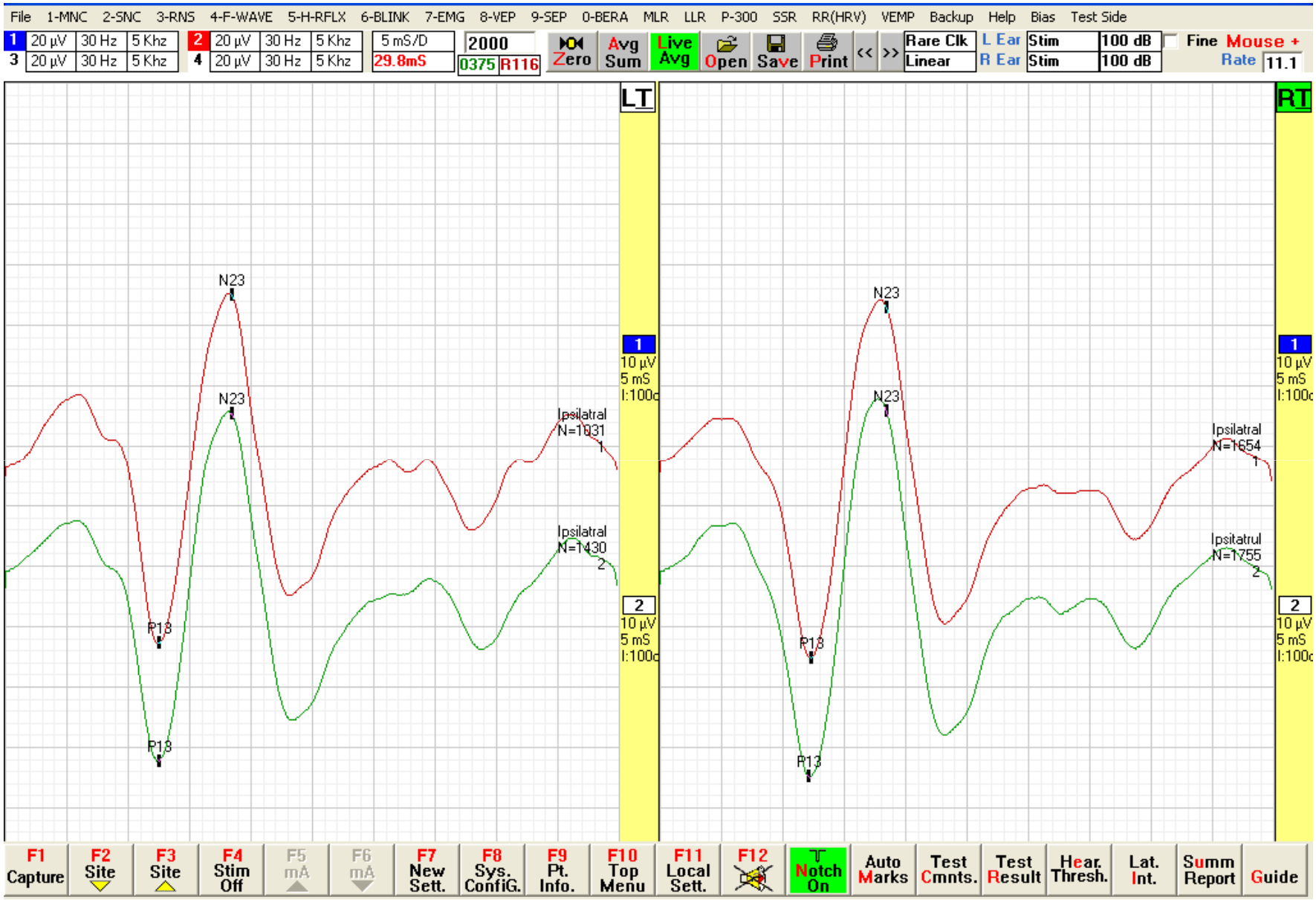

File 1-MNC 2-SNC 3-RNS 4-F-WAVE 5-H-RFLX 6-BLINK 7-EMG 8-VEP 9-SEP 0-BERA MLR LLR P-300 SSR RR(HRV) VEMP Backup Help Bias Test Side
1 20 µV 30 Hz 5 Khz
3 20 µV 30 Hz 5 Khz
2 20 µV 30 Hz 5 Khz
4 20 µV 30 Hz 5 Khz
5 mS/D
29.8mS
2000
0375 R116
Zero Avg Sum Live Avg Open Save Print << >>
Rare Clk
Linear
L Ear Stim 100 dB
R Ear Stim 100 dB
Fine Mouse +
Rate 11.1
LT
RT
N23
P13
Ipsilatral N=1931
Ipsilatral N=1430
Ipsilatral N=1654
Ipsitatrul N=1755
10 µV 5 mS I:100c
F1 Capture
F2 Site
F3 Site
F4 Stim Off
F5 mA
F6 mA
F7 New Sett.
F8 Sys. ConfiG.
F9 Pt. Info.
F10 Top Menu
F11 Local Sett.
F12
Notch On
Auto Marks
Test Cmnts.
Test Result
Hear. Thresh.
Lat. Int.
Summ Report
Guide

Table 2: Comparison of p13 and n23 latencies, p13-n23 amplitude among different age groups.

AGE GROUPS	p 13 LATENCY (ms)	n 23 LATENCY (ms)	p13 - n23 AMPLITUDE (µV)
17 – 20	11.7 ± 1.7	21.8 ± 3.3	44.14 ± 8.8
21 – 30	11.7 ± 1.5	21.7 ± 3	45.9 ± 6.1
31 – 40	11.7 ± 1	21.8 ± 2.4	1.2 ± 5.5
41 – 50	11.7 ± 1.2	21.8 ± 2.1	40.4 ± 4.6
51 – 60	11.6 ± 1	21.3 ± 1.8	30.1 ± 5.9 *
61 – 70	11.6 ± 1	21.3 ± 1.8	19.2 ± 3.2 *
Mean values	11.7 ± 1.2	21.6 ± 2.4	38.49 ± 5.7

****significant with p value <0.05.***

5.3.1. P13 LATENCY: (ms)

The mean p13 latency remained to be consistent across the age group from 17-70 years with the mean value of 11.7 ± 1.2 and no significant differences was observed between the different age groups.(Table 2, Figure 2).

5.3.2. N23 LATENCY: (ms)

The mean n23 latency were remained to be consistent across the age group from 17-70 years with the mean value of 21.6 ± 2.4 and no significant differences was observed between the different age groups.(Table 2, Figure 3)

5.3.3. P13-N23 AMPLITUDE:

The mean p13- n23 amplitude showed a significant decline ($p<0.001$) in the amplitude above 50 years of age.(Table 2, Figure 4).

Figure 2:

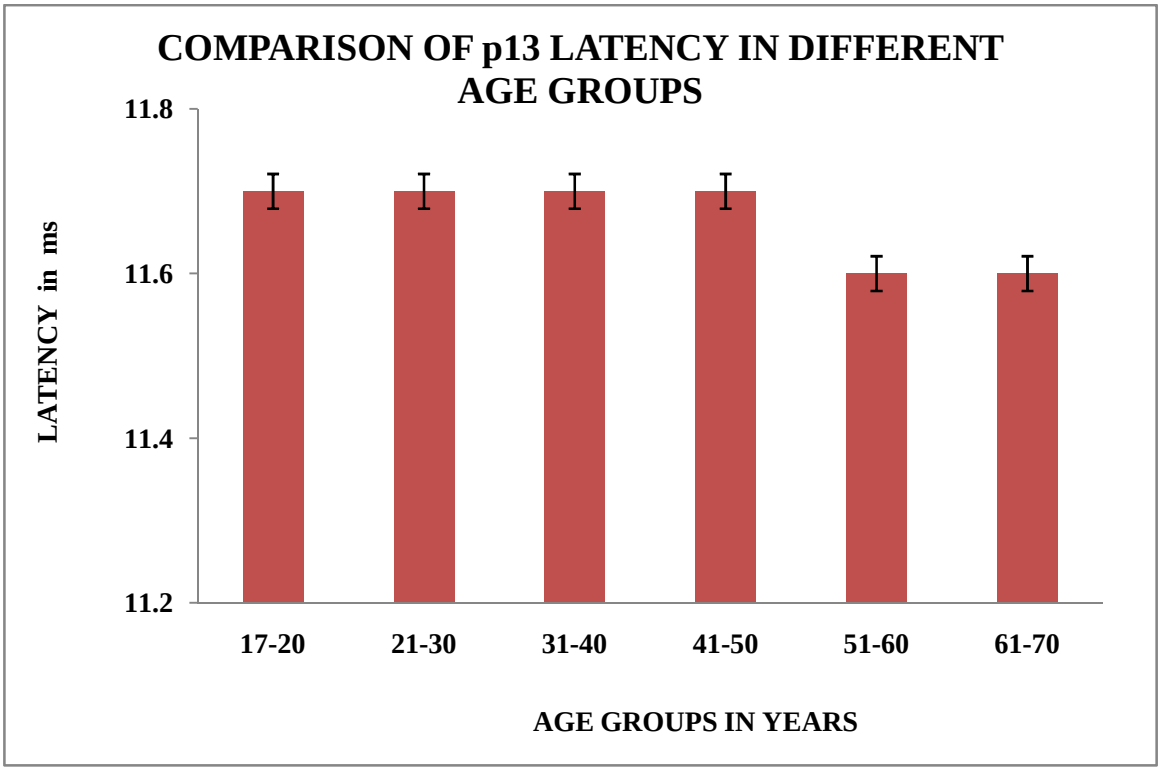


Figure 3:

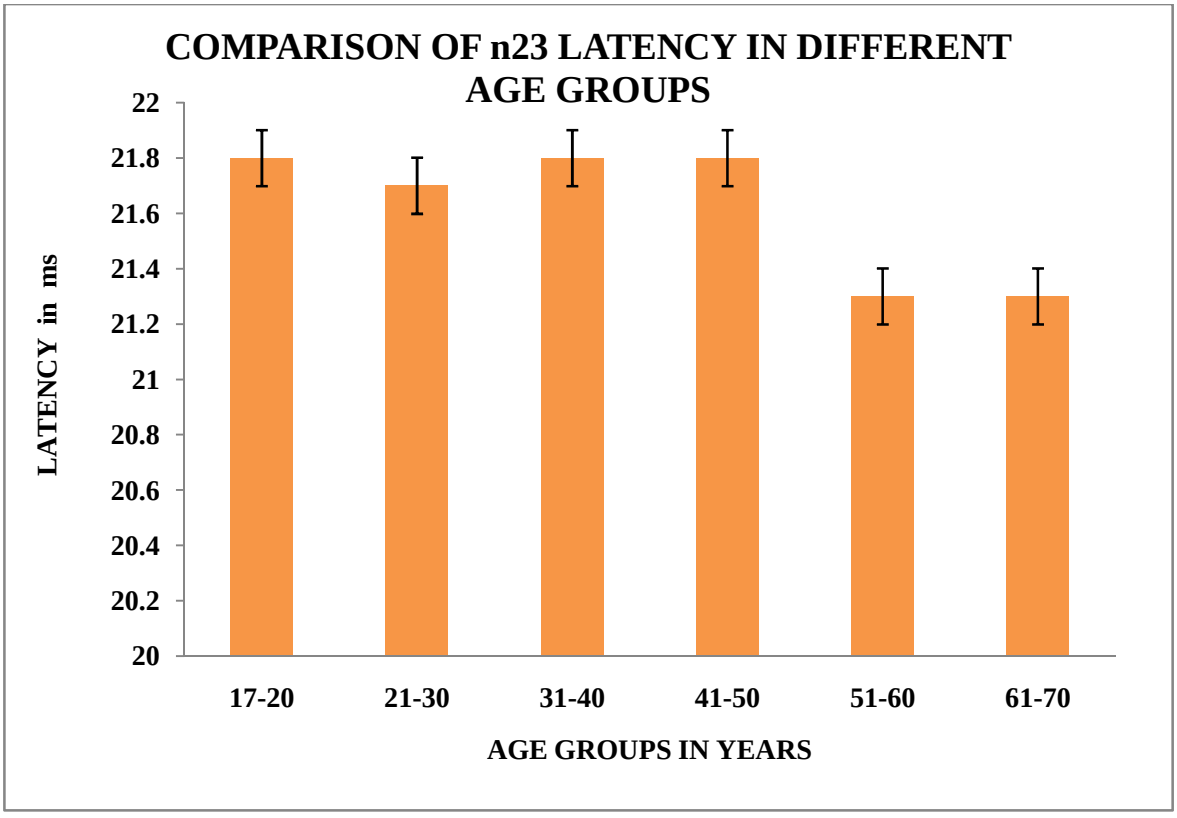
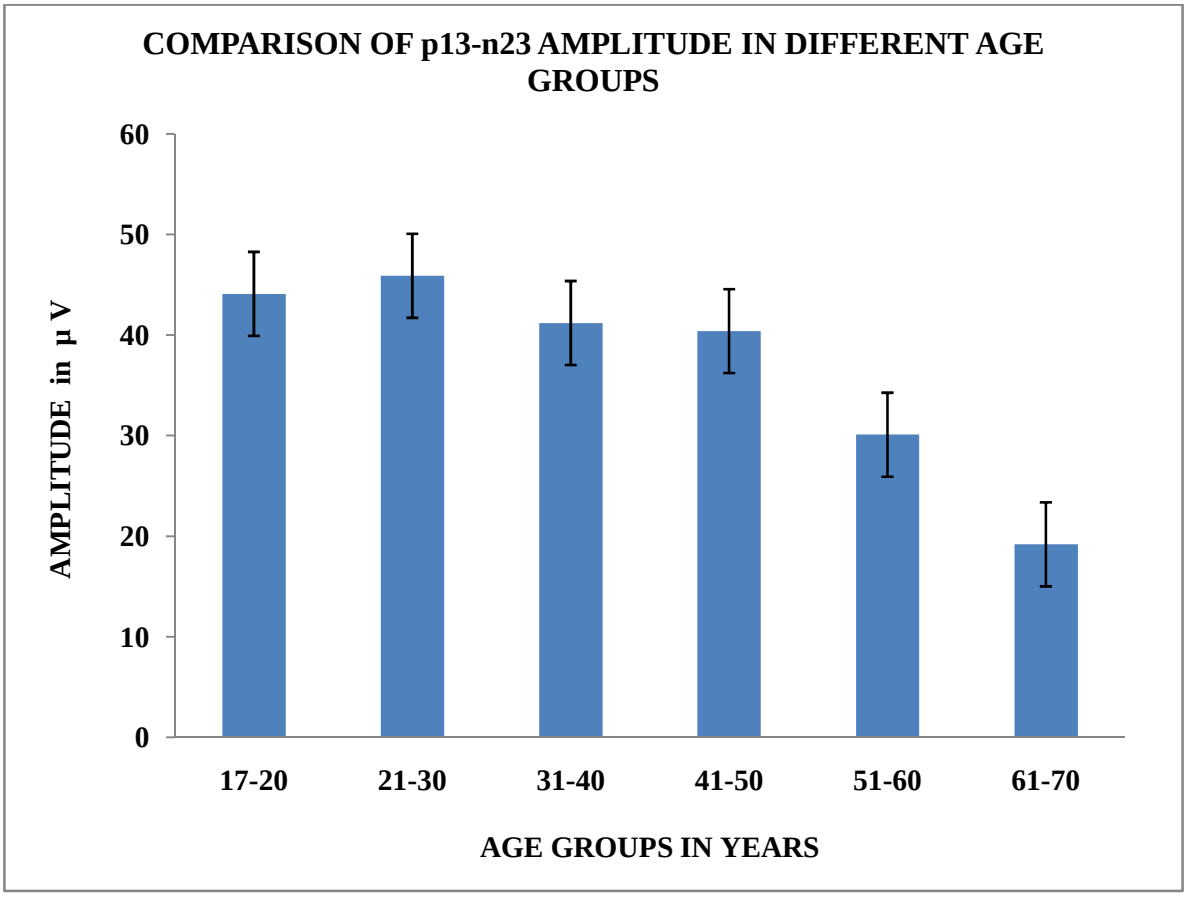


Figure 4:


COMPARISON OF p13-n23 AMPLITUDE IN DIFFERENT AGE GROUPS
AMPLITUDE in µ V
60
50
40
30
20
10
0
17-20
21-30
31-40
41-50
51-60
61-70
AGE GROUPS IN YEARS

5.2.4. INTER-AMPLITUDE DIFFERENCE RATIO (IAD %) :

The interamplitude difference ratio was calculated using the formula [(AR-AL) / (AR+AL)] X 100, where AR = amplitude of the right ear. AL = amplitude of the left ear.

The interamplitude difference ratio was higher above 50 years of age but showed no significance.(Table 3).

Table 3: Comparison of Interamplitude difference ratio during binaural (B/L) and monoaural (U/L) stimulation in different age groups.

Age Groups In Years	**IAD B/L MEAN ± SD**	**IAD U/L MEAN ± SD**	**'P' VALUE**	**IAD % B/L**	**IAD % U/L**
17 – 20	0.6 ± 5	-0.4 ± 4.1	0.3 #	8	6
21 – 30	-2 ± 5.2	1.7 ± 2.4	0.1 #	6	6
31 – 40	-0.7 ± 4.9	1.3 ± 3.1	0.1 #	8	7
41 – 50	1.4 ± 5.5	0.9 ± 5	0.5 #	9	7
51 – 60	2.1 ± 7.5	1.5 ± 6.4	0.4 #	12	11
61 – 70	4.5 ± 6.2	2.2 ± 8.1	0.2 #	16	12

 # not significant

5.4.COMPARISON OF TWO MODES OF NECK TORSION IN THE CONTROL GROUP:

25 healthy individuals of 17 to 26 years from the control group consisting of 11 males and 14 females with mean age of (20.9 ± 3.1) years has been involved for studying the comparison between two modes of neck torsion to produce reliable VEMP,

(i) Lifting head forwards towards centre

(ii) Lifting the head forward and turning to the side away from the source of stimulus.

The results were statistically analysed and their significance determined using appropriate students 't' test .

5.4.1. P13 LATENCY:

5.4.1.1. Comparison of p13 latencies between two modes of neck torsions:

Table 4: Comparison of p13 latencies between two modes of neck torsions

MODE OF NECK TORSION	RIGHT EAR p13 latency (ms)	LEFT EAR p13 latency (ms)	'p' value
Head lifted towards centre	11.8 ± 1.3	11.7 ± 1.2	0.4
Head lifted and turned away from source of stimulus	11.7 ± 1.3	11.7 ± 2	0.5
'p' value	0.4 #	0.5 #	-

 # not significant

No significant difference in mean p13 latency was observed between two modes of neck torsion for both the ears.(Table 4,Figure 5).

5.4.2. N23 LATENCY: (ms)

5.4.2.2. Comparison of n23 latencies between two modes of neck torsion:

Table 5: **Comparison of n23 latencies between two modes of neck torsion**

MODE OF NECK TORSION	RIGHT EAR n23 latency (ms)	LEFT EAR n23 latency (ms)	'p' value
HEAD LIFTED TOWARDS CENTRE	21.6 ± 2.6	21.8 ± 3.4	0.4 #
HEAD LIFTED AND TURNED AWAY FROM SOURCE OF STIMULUS	21.7 ± 1.9	21.7 ± 1.7	0.5 #
'p' value	0.4 #	0.5 #	-

 # not significant

No significant difference in mean p13 latency was observed between two modes of neck torsion for both the ears. (Table 5,Figure 6).

Figure 5:

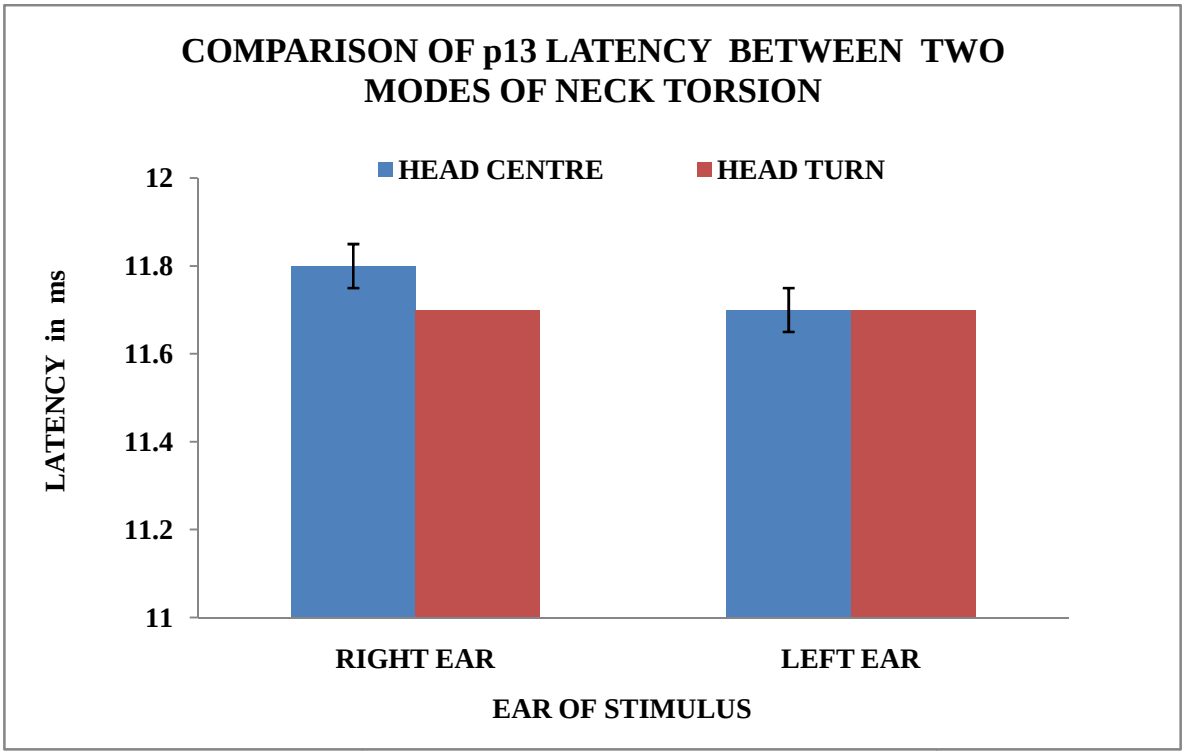
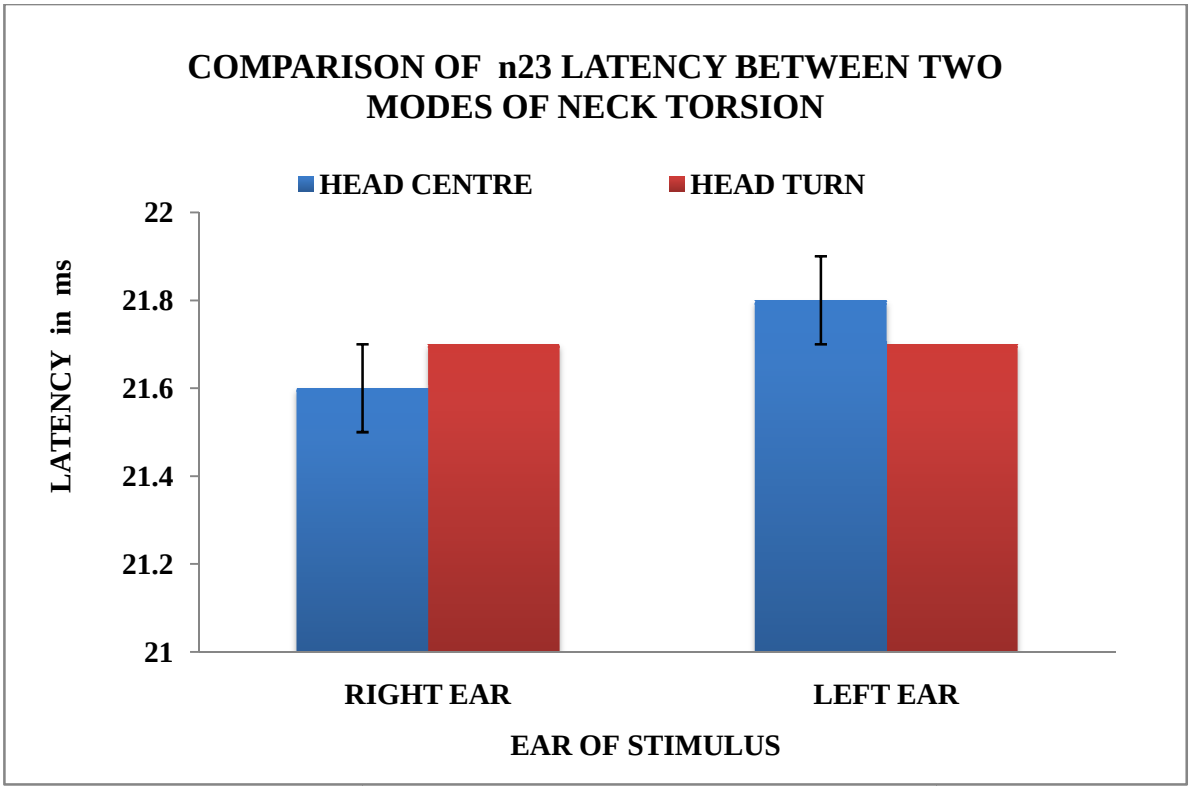


Figure 6 :



5.4.3.P13-N23 AMPLITUDE: (µV):

5.4.3.1. Comparison of p13-n23 amplitude between two modes of neck torsion:

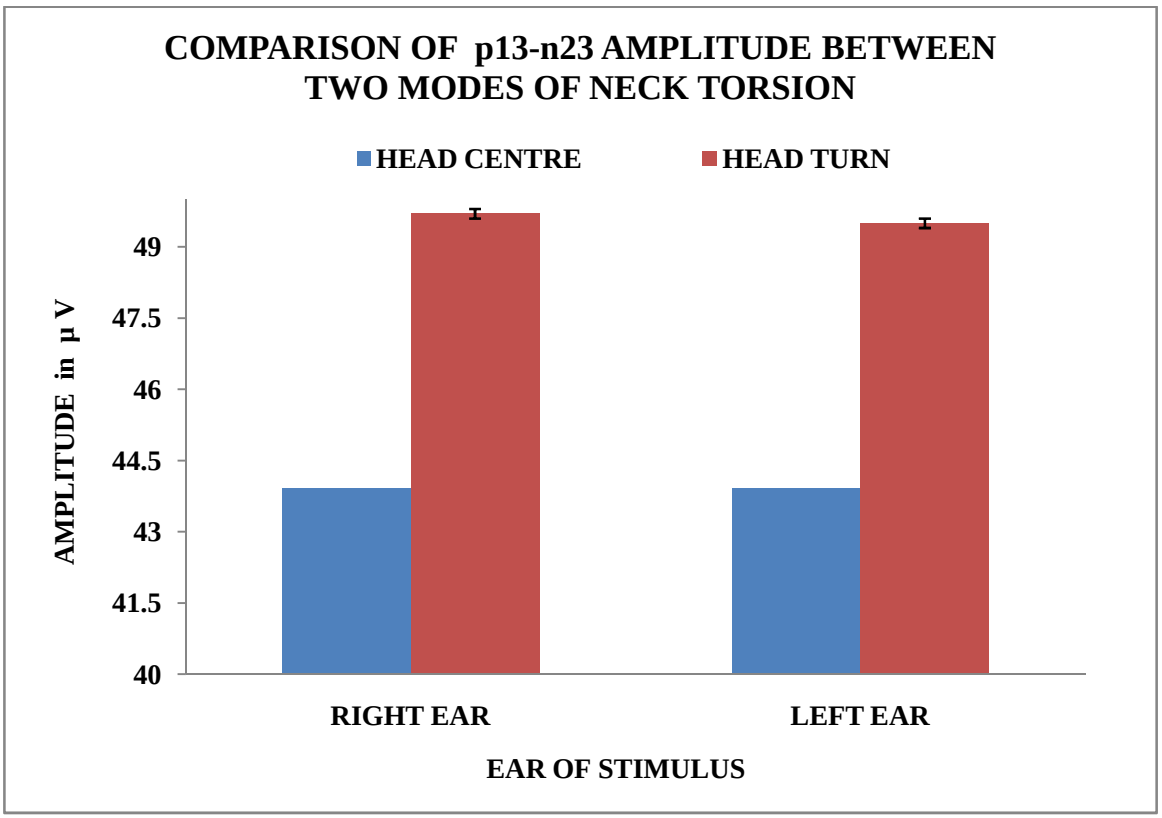
Table 6: **Comparison of p13-n23 amplitude between two modes of neck torsion.**

MODE OF NECK TORSION	RIGHT EAR p13-n23 (µV)	LEFT EAR p13-n23 (µV)	'p' value
HEAD LIFTED TOWARDS CENTRE	43.9 ± 7	43.9 ± 6.8	0.5 #
HEAD LIFTED AND TURNED AWAY FROM SOURCE OF STIMULUS	49.7 ± 7.5	49.5 ± 7.3	0.5 #
'p' value	0.003 *	0.003 *	-

 # not significant , * highly significant with p value >0.05

A highly significant increase in p13-n23 amplitude of the VEMP response was seen for both the ears while lifting head forward and turned to the side away from the source of stimulus , (p = 0.003 for right ear and p=0.003 for left ear).(Table 6,Figure 7).

Figure 7:


COMPARISON OF p13-n23 AMPLITUDE BETWEEN TWO MODES OF NECK TORSION
HEAD CENTRE
HEAD TURN
AMPLITUDE in µ V
49
47.5
46
44.5
43
41.5
40
RIGHT EAR
LEFT EAR
EAR OF STIMULUS

5.4.4. INTERAMPLITUDE DIFFERENCE RATIO :

Table 7: Comparison of Interamplitude difference ratio between two modes of neck torsion.

STUDY GROUP	IAD U/L Mean ± SD	IAD % U/L
Head towards centre	0.1 ± 3.9	6
Head turned away from stimulus	0.3 ± 3.6	7
'p' value	0.4 #	-

 # not significant

No significant change was observed in the interamplitude difference ratio between the two modes of neck torsion.(Table 7).

5.5. COMPARISON OF THE DIFFERENT PLACEMENTS OF INVERTING/ REFERENCE ELECTRODE IN THE CONTROL GROUP.

20 healthy individuals in the age group of 17-26 years from the control group (mean 21±3.1) were assessed for the influence of inverting electrodes on the VEMP waveforms. The reference or inverting electrodes were placed at different sites such as sternum, mastoid and the wrist; and the VEMP response were statistically analysed and their significance derived using one way ANOVA followed by Tukeys multiple comparison tests.

5.5.1. P13 LATENCY:

The p13 latency during binaural stimulation of the VEMP response showed no significant difference (df=2, F=3.96 for right, df=2, F=8.5 for left) when reference electrodes were placed at the sternum, mastoid or the wrist . (Table 8,Figure 8).

The p13 latency during monoaural stimulation of the VEMP response showed no significant difference (df=2, F=4.1 for right, df=2, F=4.3 for left) when reference electrodes were placed at the sternum, mastoid or the wrist. (Table 8,Figure 8).

No significant change was observed during binaural and monoaural stimulation.

Table 8: Comparision of p13 latency among different placement of electrodes

P13 LATENCY	Sternum	Wrist	Mastoid
B/L LT	12.9±0.7	11.9±0.9	12.1±0.7
B/L RT	12.5±1.5	11.7±0.8	11.7±0.8
RT	12.9±1.2	11.6±2.2	11.9±1.1
LT	12.7±0.9	12.1±0.9	12.2±0.7

5.5.2. N23 LATENCY:

The n13 latency during binaural stimulation of the VEMP response showed no significant difference (df=2, F=4.04 for right, df=2, F=2.9 for left) when reference electrodes were placed at the sternum, mastoid or the wrist . (Table 9,Figure 9).

The n13 latency during monoaural stimulation of the VEMP response showed no significant difference (df=2, F=1.6 for right, df=2, F=4.945 for left) when reference electrodes were placed at the sternum, mastoid or the wrist. (Table 9,Figure 9).

No significant change was observed during binaural and monoaural stimulation.

Table 9:Comparision of n23 latency among different placement of electrodes

N23	Shernum	wrist	Mastoid
B/L LT	22.3±1.7	20.9±2.2	21.7±1.7
B/L RT	22.5±2.5	20.2±3.2	21.9±2.8
RT	22±2.3	20.9±1.8	21.3±2.3
LT	22.6±1.5	20.4±3.2	21.5±2.2

5.5.3. P13-N23 AMPLITUDE:

The p13 latency during binaural stimulation of the VEMP response showed no significant difference (df=2, F=6.6 for right, df=2, F=7.6 for left) when reference electrodes were placed at the sternum, mastoid or the wrist . (Table 10,Figure 10).

The p13 latency during monoaural stimulation of the VEMP response showed no significant difference (df=2, F=7.7 for right, df=2, F=4.2 for left) when reference electrodes were placed at the sternum, mastoid or the wrist. (Table 10,Figure 10).

A significant increase ($p<0.001$) in amplitude was found when the inverting electrode was placed in the sternum compared to that of the wrist or mastoid.

Table 10: Comparison of p13-n13 amplitude among different placement of electrodes.

P13-n23amplitude	Shernum	Wrist	Mastoid
B/L LT	53.4±4.1	51.1±4.8	51.9±3.6
RT	55.2±4.6	50.5±4.7	52.1±4.2
RT	54.9±4.1	49.9±4.5	51.6±4.3
LT	55.1±5	51.2±4.3	52.1±5

5.5.4. INTERAMPLITUDE DIFFERENCE RATIO:

Table 11: Comparison of interamplitude difference ratio among different placement of electrodes.

STUDY GROUP	IAD B/L Mean ± SD	IAD U/L Mean ± SD	IAD % B/L	IAD % U/L
sternum	-0.1 ± 1.5	-0.1 ± 3	6	2
wrist	-0.5 ± 1.6	-1.3 ± 2.5	5	3
mastoid	0.2 ± 1.5	-0.5 ± 2	4	4

No significant change was observed in the interamplitude difference ratio among different placement of electrodes.

Figure 8:

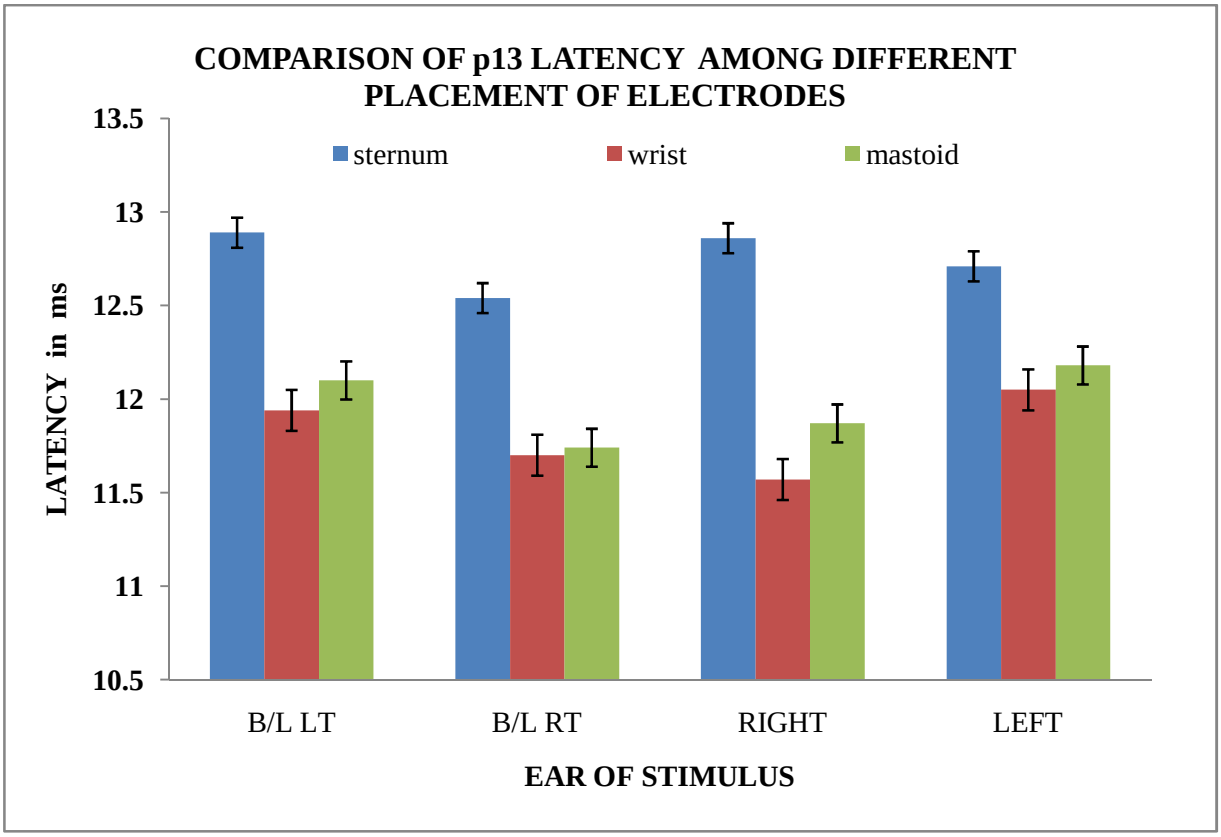


Figure 9:

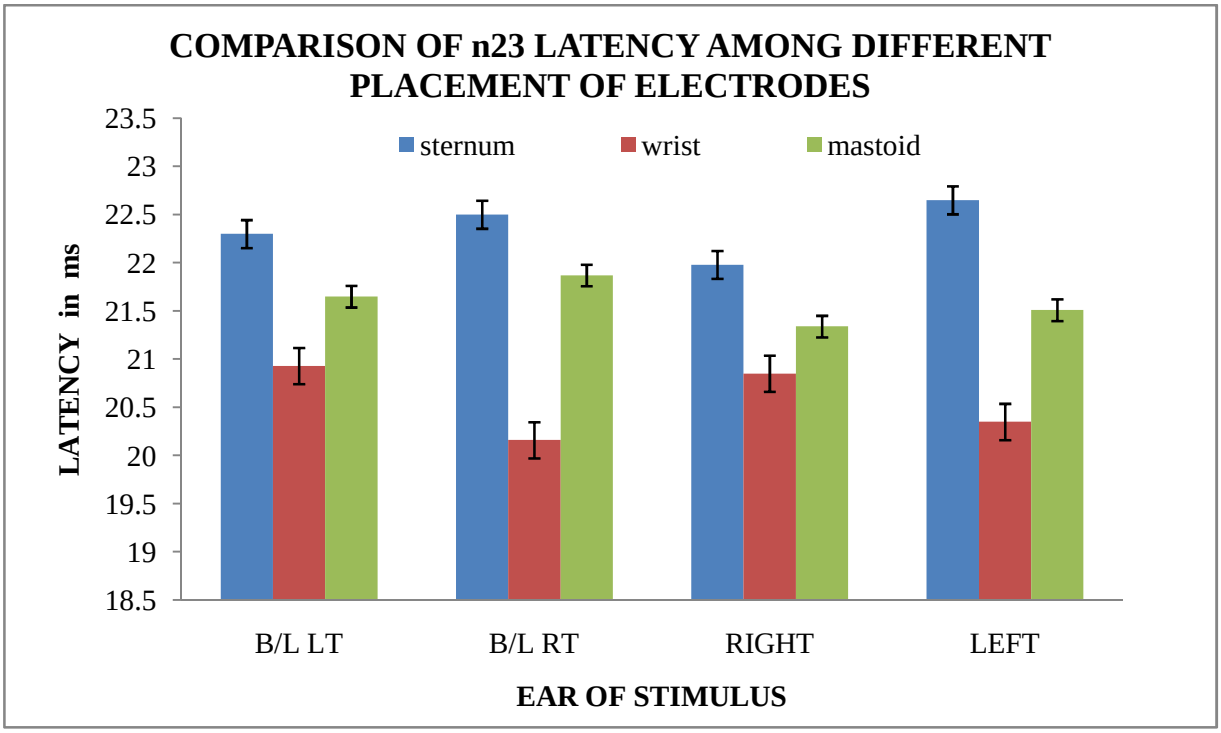
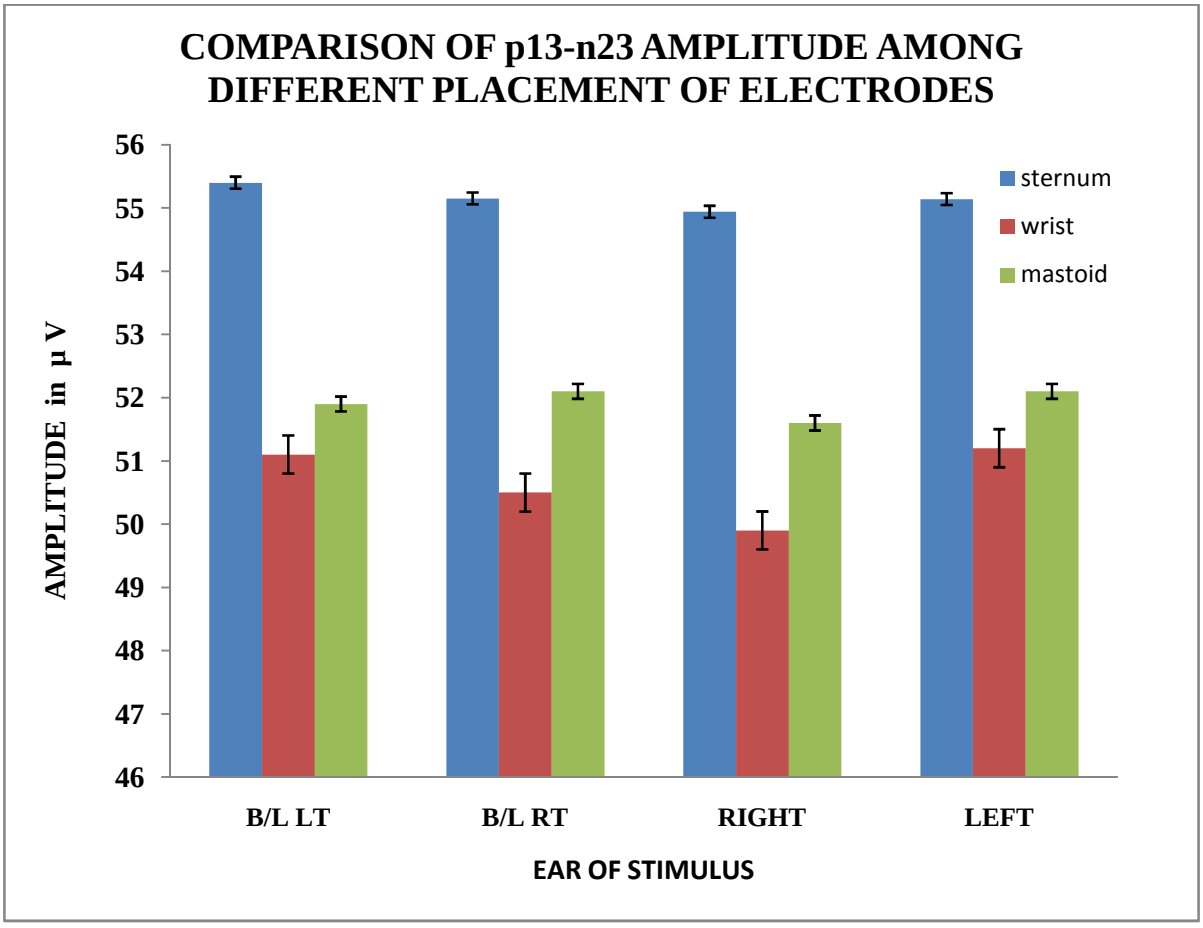


Figure 10:


COMPARISON OF p13-n23 AMPLITUDE AMONG DIFFERENT PLACEMENT OF ELECTRODES
AMPLITUDE in µ V
56
55
54
53
52
51
50
49
48
47
46
sternum
wrist
mastoid
B/L LT
B/L RT
RIGHT
LEFT
EAR OF STIMULUS

5.6.COMPARISON OF MIGRAINE PATIENTS WITH AND WITHOUT VERTIGO WITH AGE AND SEX MATCHED CONTROLS :

As per the ICHS-2 , the study group of 50 migraine patients were further classified into two groups

1. those with migraine alone (n=20) and
2. those who had vertigo associated with migraine (n=30).

The results were statistically analysed for their significance using appropriate students t test.

5.6.1.MIGRAINE PATIENTS WITHOUT VERTIGO COMPARED WITH AGE-SEX MATCHED CONTROLS.

20 patients had definite migraine without vertigo were compared with age and sex matched controls. Unpaired students ‘t’ test used to compare the mean latencies and amplitudes of VEMP between the control and the migraine patients.

5.1.1. P13 LATENCY : (ms)

TABLE 12: Comparison of p13 latency between the control and the migraine patients without vertigo.

P13 latency (ms)	Bilateral left Mean±SD	Unilateral left Mean ±SD	'p' value	Bilateral right Mean±SD	Unilateral right Mean ±SD	'p' value
Control group n=50	11.84 ± 2.2	11.82 ± 1.4	0.5	11.8 ± 1.6	11.94 ± 1.2	0.4#
Migraine patients n=50	11.73 ± 1	11.7 ± 1.1	0.5	11.83 ± 1.5	11.94 ± 1.1	0.4#
'p' value	0.4#	0.4#	-	0.5#	0.5#	-

 # not significant

There was no statistically significant difference in p13 latency observed between the control and migraine patients without vertigo. (Table 12,Figure 11).

No significant difference in p13 latency between binaural and monoaural stimulation was found between controls and migraine patients.(Table 12,Figure 11).

5.6.1.2.N23 LATENCY : (ms)

Table 13 : Comparison of n23 latency between the control and migraine patients without vertigo.

N23 latency (ms)	Bilateral left Mean±SD	Unilateral left Mean ±SD	'p' value	Bilateral right Mean±SD	Unilateral right Mean ±SD	'p' value
Control group n=50	20.4 ± 2.6	21.7 ± 2.2	0.06	21.8 ± 2.3	20.8 ± 1.4	0.07 #
Migraine patients n=50	20.8 ± 2.6	21.8 ± 3	0.1	21.7 ± 1.6	20.9 ± 2.1	0.08 #
'p' value	0.3 #	0.4 #	-	0.5 #	0.5 #	-

 # not significant

There was no statistically significant difference in n23 latency observed between the control and migraine patients without vertigo.

No significant difference between binaural and monoaural stimulation was found between controls and migraine patients without vertigo.(Table 13, Figure 12.).

Figure 11:

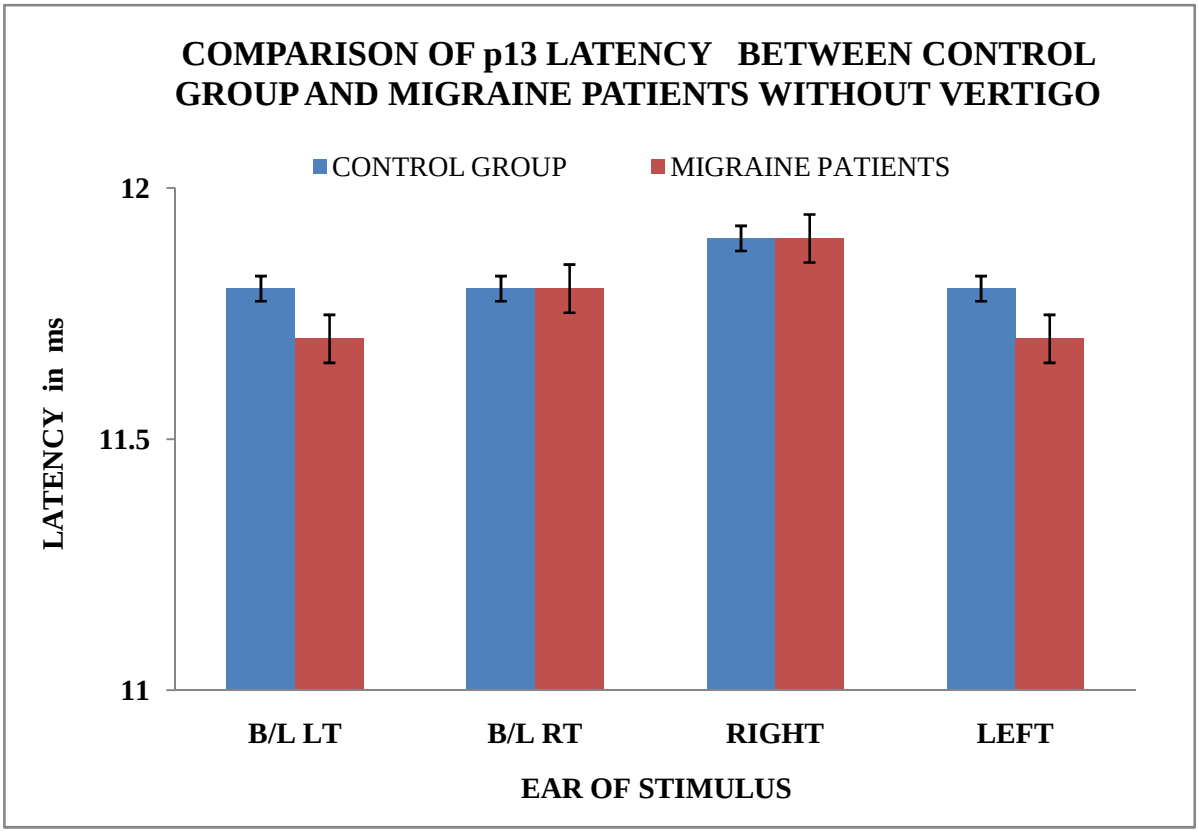
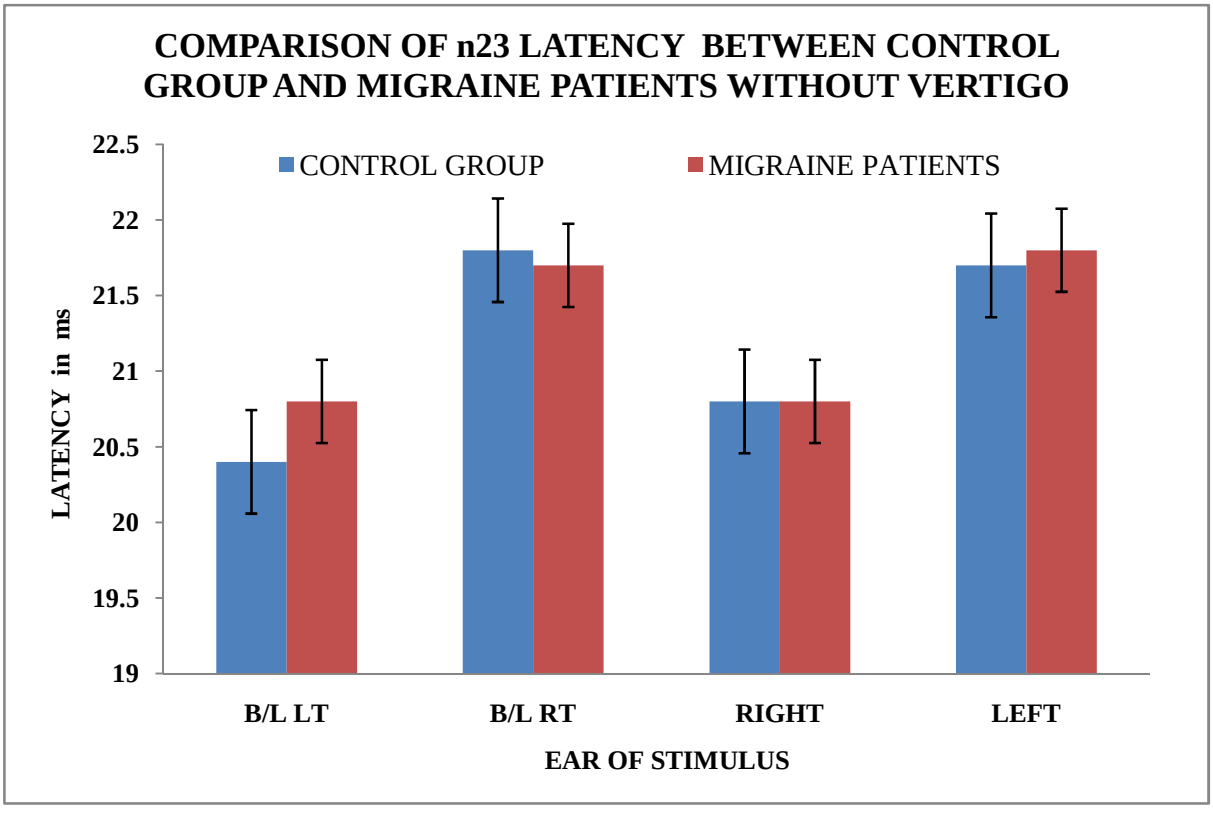


Figure 12:



5.6.1.3.P13-N23 AMPLITUDE :

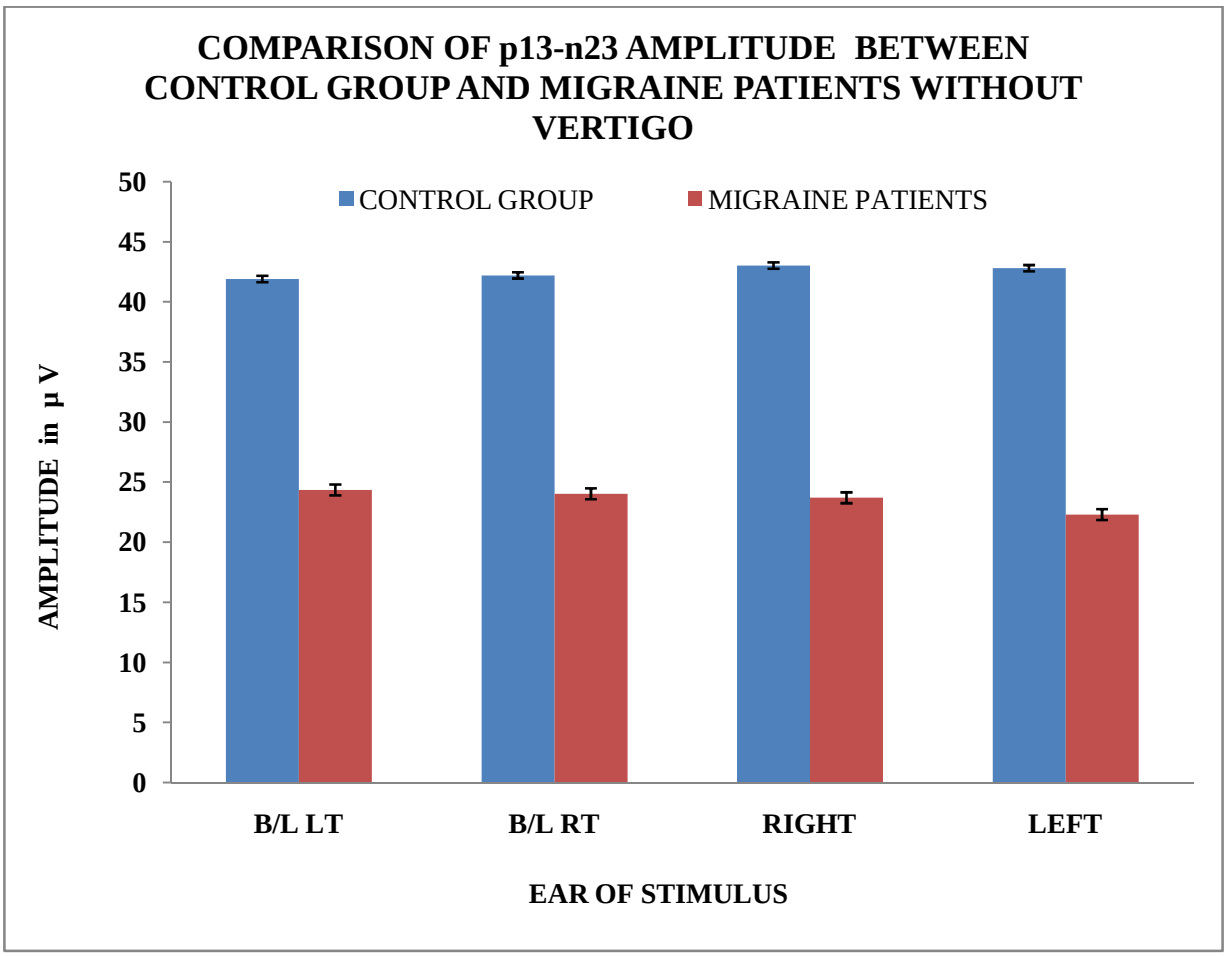
Table 14: Comparison of p13-n23 amplitude between the control and the migraine patients without vertigo.

p 13 - n23 amplitude (µV)	**Bilateral left Mean±SD**	**Unilateral left Mean ±SD**	**'p' value**	**Bilateral right Mean±SD**	**Unilateral right Mean ±SD**	**'p' value**
Control group n=50	42 ± 6.4	42.8 ± 8	0.4 #	42.2 ± 4.7	43.02 ± 6.8	0.3 #
Migraine patients n=50	24.4 ± 8.1	22.3 ± 6.6	0.2 #	24.03 ± 8.5	23.7 ± 7.8	0.4 #
'p' value	1.6E-09 *	3.93E-11 *	-	1.77E-10 *	1.79E-10 *	-

 # not significant ; * highly significant p value <0.05.

A highly significant decrease in amplitude was found between the migraine patients without vertigo and the control group ($p<0.001$) both during binaural stimulation ($p=1.6E-09$ for left ear, $p=1.77E-10$ for right ear) and monoaural stimulation ($p=1.79E-10$ for right ear, $p=3.93E-11$ for left ear). (Table 14,Figure 13).

Figure 13:


COMPARISON OF p13-n23 AMPLITUDE BETWEEN CONTROL GROUP AND MIGRAINE PATIENTS WITHOUT VERTIGO
CONTROL GROUP
MIGRAINE PATIENTS
AMPLITUDE in µ V
50
45
40
35
30
25
20
15
10
5
0
B/L LT
B/L RT
RIGHT
LEFT
EAR OF STIMULUS

5.6.1.4.IAD RATIO (%) :

Table 15: Comparison of IAD ratio between the control and the migraine patients without vertigo.

STUDY GROUP	IAD binaural stimulus Mean ± SD	IAD monoaural stimulus Mean ± SD	IAD % binaural stimulus	IAD % monoaural stimulus
Controls	0.6 ± 5	0.5 ± 5.4	12	7
Migraine patients without vertigo	-0.6 ± 14.2	2.5 ± 8.6	19	14
'p' value	0.3 #	0.1 #	-	-

 # not significant

The interamplitude difference ratio during binaural stimulation was 19% in the migraine patients without vertigo and 12 % in the control group. No significance was observed for the increased IAD% in migraine patients.

The interamplitude difference ratio during monoaural stimulation was 14% in the migraine patients without vertigo and 7 % in the control group. No significance was observed for the increased IAD% in migraine patients. (Table 15).

5.7.MIGRAINE PATIENTS WITH VERTIGO COMPARED WITH AGE-SEX MATCHED CONTROLS.(N=30).

30 migraine patients with vertigo were compared with age and sex matched controls. Unpaired students 't' test was used to compare the mean latencies and amplitudes of VEMP between the control and the migraine patients.

5.7.1.P13 LATENCY (ms):

Table 16: Comparison of p13 latency between the control and the migraine patients with vertigo.

P13 latency (ms)	Bilateral left Mean±SD	Unilateral left Mean ±SD	'p' value	Bilateral right Mean±SD	Unilateral right Mean ±SD	'p' value
Control group n=30	11.8 ± 2.2	11.9 ± 1.3	0.4 #	11.9 ± 1.7	11.9 ± 1.3	0.5 #
Migrainous vertigo patients n=30	11.9 ± 1.7	11.9 ± 1.3	0.5 #	11.9 ± 1.4	11.8 ± 2.5	0.4 #
'p' value	0.4 #	0.5 #	-	0.4 #	0.4 #	-

 # not significant

There was no statistically significant difference in p13 latency between the controls and migraine patients with vertigo. (Table 16,Figure 14).

5.7.2.N23 LATENCY : (ms)

Table 17: Comparison of n23 latency between the control and the migraine patients with vertigo.

n23 latency (ms)	**Bilateral left Mean±SD**	**Unilateral left Mean ±SD**	**'p' value**	**Bilateral right Mean±SD**	**Unilateral right Mean ±SD**	**'p' value**
Control group n=50	21.8 ± 3.3	21.9 ± 2.6	0.4 #	21.9 ± 2.8	21.7 ± 2.6	0.4 #
Migraine patients n=50	21.8 ± 2.4	21.7 ± 4.7	0.5 #	21.7 ± 2.6	21.8 ± 2.4	0.5 #
'p' value	0.5 #	0.4 #	-	0.4 #	0.5 #	-

 # not significant

There was no statistically significant difference in n23 latency between the controls and migraine patients with vertigo.(Table 14,Figure 15).

Figure 14:

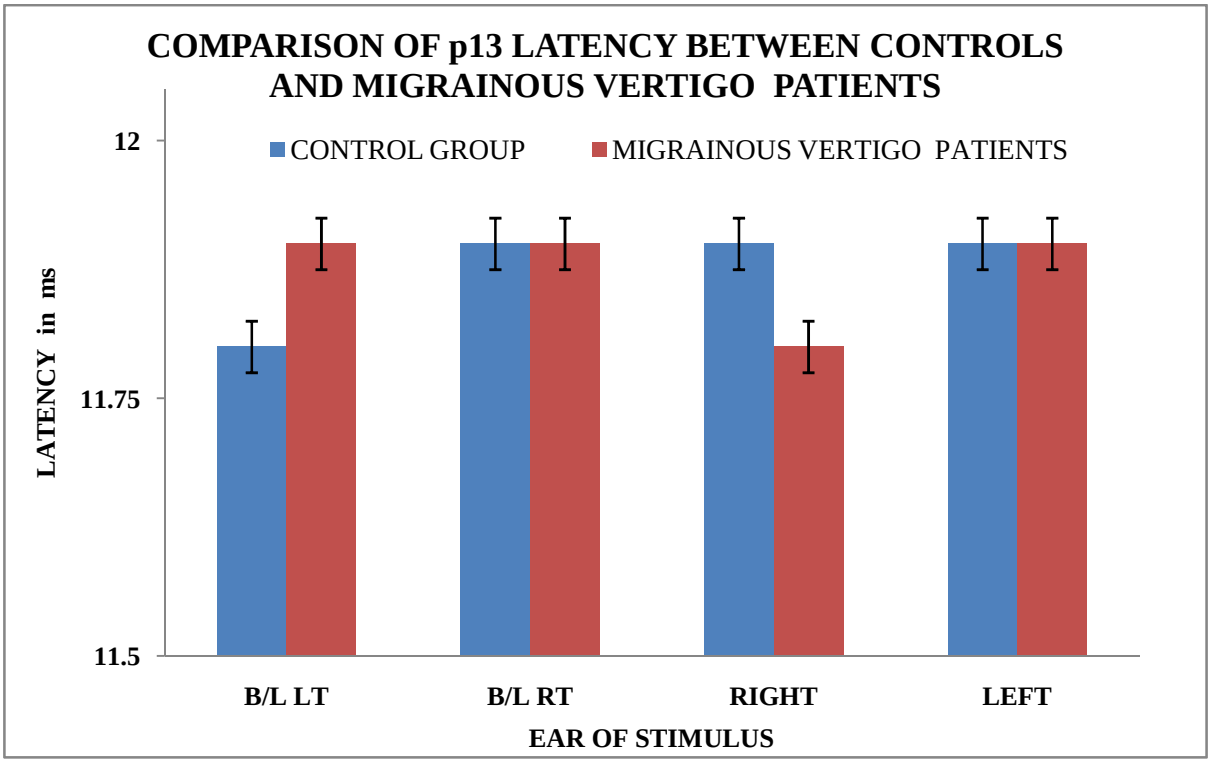
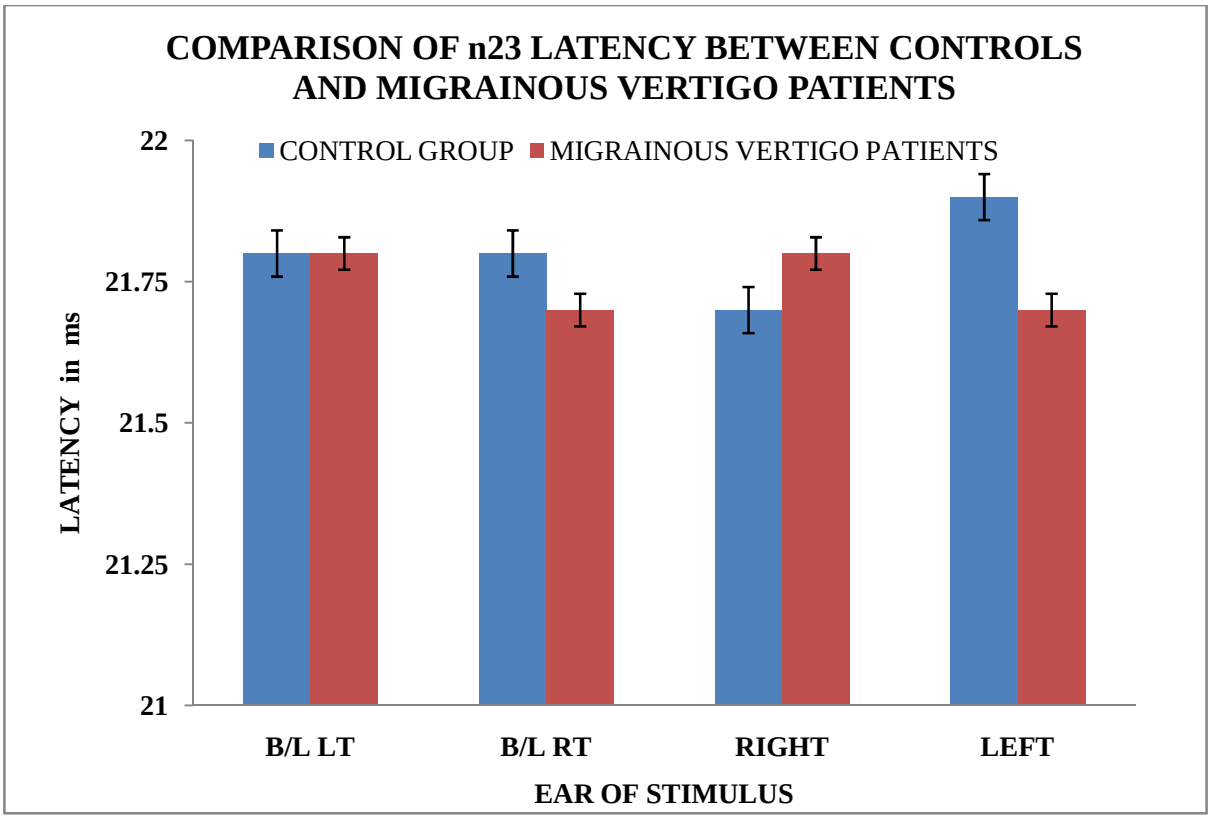


Figure 15:



5.7.3.P13-N23 AMPLITUDE :

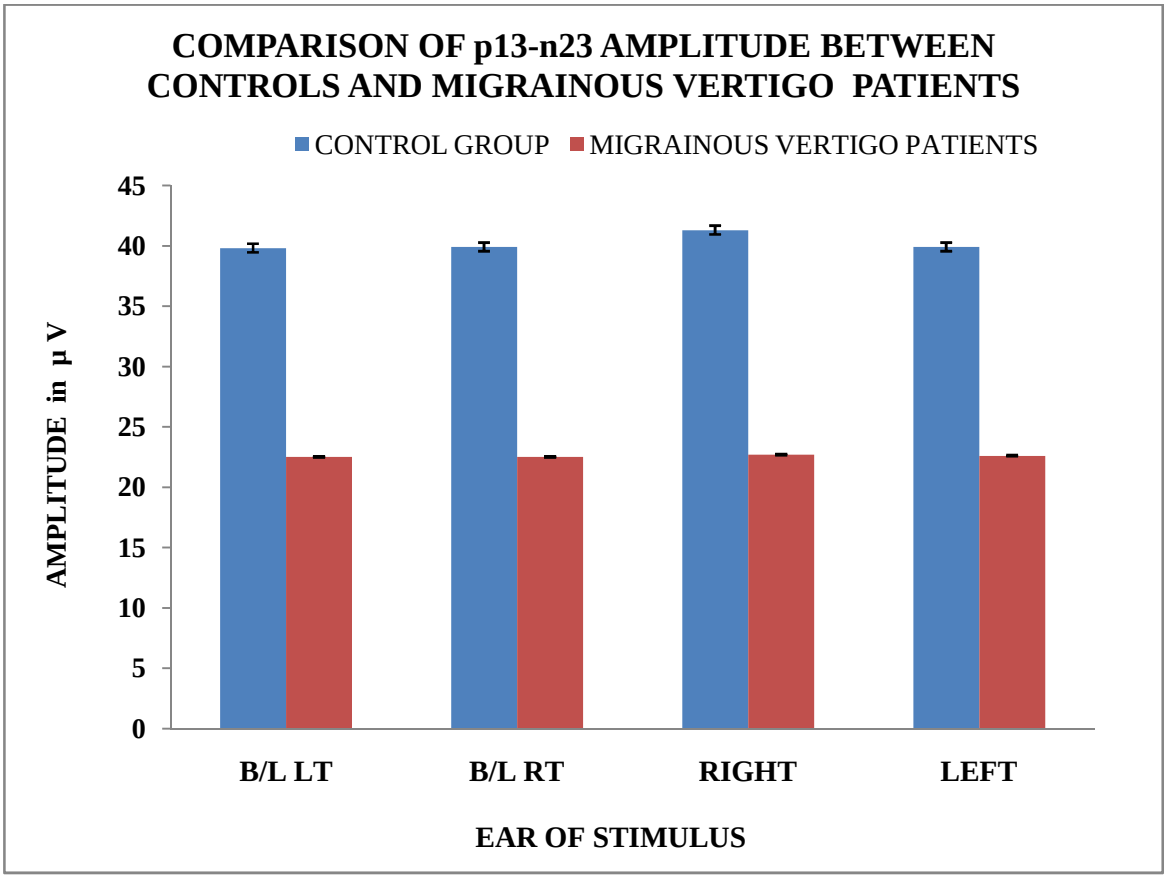
Table 18 : Comparison of p13-n23 amplitude between the controls and the migraine patients with vertigo.

p 13 - n23 amplitude (µV)	**Bilateral left Mean±SD**	**Unilateral left Mean ±SD**	**'p' value**	**Bilateral right Mean±SD**	**Unilateral right Mean ±SD**	**'p' value**
Control group n=50	39.7 ± 9.2	39.9 ± 8	0.5	39.9 ± 9.5	41.3 ± 8.9	0.3 #
Migraine patients n=50	22.5 ± 7.5	22.5 ± 7.1	0.5	22.5 ± 7.2	22.7 ± 8.4	0.5 #
'p' value	3.13E-11 *	8.12E-13 *	-	2.78E-11 *	1.72E-12 *	-

 # not significant, * highly significant with p value >0.05

A highly significant decrease ($p<0.001$) was found between the control group and the migraine patients with vertigo (Table 15,Figure 16).

Figure 16:


COMPARISON OF p13-n23 AMPLITUDE BETWEEN CONTROLS AND MIGRAINOUS VERTIGO PATIENTS
CONTROL GROUP
MIGRAINOUS VERTIGO PATIENTS
AMPLITUDE in µ V
45
40
35
30
25
20
15
10
5
0
B/L LT
B/L RT
RIGHT
LEFT
EAR OF STIMULUS

5.7.4. INTERAMPLITUDE DIFFERENCE RATIO %:

Table 19: Comparison of interamplitude difference ratio between the controls and migraine patients with vertigo.

STUDY GROUP	IAD B/L Mean ± SD	IAD U/L Mean ± SD	IAD % B/L	IAD % U/L
Controls	0.03 ± 8.7	1.4 ± 4.8	16	11
Migraine patients with vertigo	0.3 ± 9	-0.3 ± 9	17	11
'p' value	0.5 #	0.2 #	-	-

 # not significant

The interamplitude difference ratio during binaural stimulation was 17% in the migraine patients with vertigo and 16 % in the control group and no significant change was observed.(Table 19).

The interamplitude difference ratio during monoaural stimulation was 11% in the migraine patients with vertigo and 11 % in the control group and no significant change was observed. (Table 19).

6. DISCUSSION

6. DISCUSSION

Vestibular evoked myogenic potential (VEMP) test is an useful tool in determining whether the saccule, inferior vestibular nerve and central connections are intact and working normally. The saccule which is an organ of hearing in lower animals has a slight sound sensitivity and this is measured in VEMP testing.

[141]Sound stimulates the saccule which activates the inferior nerve, lateral vestibular nucleus , 11th cranial nerve nucleus and then the ipsilateral SCM mostly through the medial vestibulospinal tract. The main source of VEMP is saccule[141]. This study on VEMP was performed on migraine patients and compared with age and sex matched controls.

1. The controls were studied for the following:

   A.) effect of age on VEMP response.

   B.) Effect of two different modes of neck torsion on VEMP response.

   C.) Effect of placement of reference/inverting electrode at different sites namely sternum, mastoid and wrist on VEMP response.

2. VEMP response on migraine patients with or without vertigo were then compared with age and sex matched controls.

6.1 SOUND STIMULUS AND PRESENTATION:

To get a VEMP response loud clicks of 80-120dB were repetitively presented to each ear in turn at 20msec intervals (5/sec) with an optimum frequency of 500Hz using sternum as reference and the forehead as the ground. The myogenic potential are amplified , band pass filtered (30-3KHZ) and analyzed for 200 presentations. The response evoked from the mid portion of SCM are averaged and presented as VEMP. The latencies p13 , n23 and p13-n23 amplitudes and interamplitude difference are to be measured.

VEMP can also be elicited by tone bursts of 500 or 1000hz at 5/sec rate of 120dB SPL. The advantage of tone bursts is that it requires lower stimulus intensities[107] .

In our study we have used loud clicks of 100 dB which were averaged for 200 presentations with a minimum of two repetitions to elicit a VEMP response using RMS multichannel polyrite.

6.2.BINAURAL / MONOAURAL STIMULATION OF VEMP:

VEMP can be elicited by binaural and/or monoaural stimulation. There are different schools of thought regarding the type of stimulus to be used.

[141]"Monoaural stimulation produces a healthy response compared to binaural since VEMPs are generally ipsilateral" because binaural stimulation may produce a "volume conduction" artefact when the electrical activity

crosses the midline. Binaural stimulus is faster but it reduces the ability to localize the side of lesion due to cross over unlike the work done by yang et al[142].

In our study we have used both binaural and monoaural stimulation and the elicited VEMP response did not show much variation.

6.3. LATENCIES p13 AND n23 (ms):

The latencies of p13 and n23 in our study did not significantly vary with regard to the different types of neck torsion or to the placement of the inverting electrodes. Similarly the latencies did not alter either with age or with the study group namely the migraine patients. As per [141]timothy et al the usefulness of measuring latencies is of less significance since it is less reliable. The only usefulness it has, is in deciding whether a particular waveform is a 'VEMP' or just noises.

People with fat neck have lower responses as the signal from the muscle is underneath the fat and has to go a longer distance. Similarly people with longer neck have later responses as the signal again has to go a longer distance[104].

Prolonged latency of VEMP indicateretrocochlear lesions such as vestibular neuritis. Abnormal asymmetrical or long latencies were also reported by [143]Murofuschi et al 1996 in about 25% of patients with vestibular neuritis.

Prolonged latencies are also seen in persons with long neck . Sometimes 'VEMP' like potentials that is caused by posterior auricular muscles (PAM) occur at a latency of 11ms which might overlap VEMP or it might be due to volume conduction. This can be ruled out by running VEMP initially or inbetween other runs without contracting SCM[144,145].

6.4. AMPLITUDE (μV) :

The most reliable parameter of the VEMP response is the amplitude[121]. The limits of normal for the amplitude for the head rising technique are roughly between 70-700[141]. Except persons with hyperacussis who shows longer amplitude , most of the diseases are diagnosed with values lesser than the upper limit.

In our study the amplitude were within the normal range in controls as well as in migraine patients. The mode of neck torsion and the placement of reference electrode or the age had no bearing on the amplitude regarding the normal range. However significant increase in the amplitude was seen when the neck was lifted forwards and the neck turned away from the source of stimulus. Similarly a significantly larger amplitude was seen when the reference electrode was placed on the sternum compared to the wrist or the mastoid .With regard to age there was a significant drop in the amplitude after 50 years of age in our study.

Other mode of neck torsion where the head is being actively turned to one side produces smaller potentials and are less reliable[146]. Some experts on their personal communication has recommended simply tilting the entire body up by 30 degrees but there are not much references for this procedure regarding amplitude[147].

[120]With regard to placement of reference /inverting electrode, li et al 1999 had advocated wrist as the better choice for eliciting a good amplitude. Devon hale identified mastoid for the same.

Regarding the influence of age in VEMP [120]su et al 2004 showed decreased amplitude (roughly a factor of 2) in persons of 70 years and older compared to younger individuals. [148]Ochi also reported amplitude of approximately 250 for younger age group and 90 for 80 years old. Presently it appears that VEMP amplitude declines as age advances. This decline is being much more in the elderly due to the physiological process of aging resulting in conductive hearing loss either due to otosclerosis or other related disorders. Even a small amount of hearing loss such as 10-15dB do not produce VEMP as reported by wang and yang 2007[128]. They attributed this effect to saccular damage. Similarly [149]zhou et al reported a reduction in VEMP response in children with sensorineural hearing loss probably due to saccular damage whereas [150]wu and yang 2002 identified normal VEMP in sudden hearing loss.

6.5.VEMP AND MIGRAINE:

As per the study by [151]Lia LJ et al 10 of the 20 patients with migraine had normal VEMP response during binaural stimulation, 7 of 20 showed absent VEMP response and 2 patients had delayed VEMPs. The author assumed the delayed and the absent VEMP are associated with brainstem lesions affecting the saccular pathways. whereas in our study no absent VEMPs were recorded and there was no significant differences between the latencies during binaural and monoaural stimulation. As per [109] Welgampola et al VEMP latencies were delayed in brainstem disorders but not in peripheral dysfunction whereas amplitudes depends rather on peripheral vestibular dysfunction.

Our data agree with the reports of [152]alen et al who stated that patients with migraine (without any vestibular symptoms) has VEMPs with low amplitudes. The author suggested that this change could be due to abnormal serotoninergic modulation or hypoperfusion induced ischemia to the vestibular nuclei.

The reduced mean amplitude in migraine does not favour vestibular hyperexcitability as an explanation for the habituation deficit in migraine but rather an abnormal processing of repeated stimuli in the reflex circuit.

According to the author marziyeh et al 2010 the prolonged latency of VEMP during the inter-attack intervals , even in the absence of vestibular

symptoms are suggestive of vestibular dysfunction in migraine patients. They also reported that there was involvement of vestibulo-spinal tract in migraine patients.

In another study prolonged latency was reported in multiple sclerosis which were probably due to demyelination[153] suggesting that latencies reflect the central vestibular processing.

The question arises as to whether the reduced amplitudes in migrainousvertigo is due to peripheral or central origin. Functional MRI studies during human migraine aura suggest that an initial cortical hyperperfusion is followed by a longer lasting wave of hypoperfusion[154,155]. Hyperperfusion induced cortical spreading depression (CSD) is assumed to be the pathophysiologic correlate of migraine aura. Blockage of nitric oxide synthesis and high levels of extracellular potassium prevents CSD hyperperfusion thus inducing CSD hypoperfusion that leads to infarction[156]. The origin of CSD was found to be from brainstem[157] suggesting that vertigo symptoms might be related to hypo-perfusion induced ischemia of labrynthine structures[158]. Ischemia of the inner ear structures would reflect as reduced VEMP amplitude as its electrophysiologic correlate.

Moreover many studies have reported 25% prevalence of peripheral vestibular abnormalities in migraine patients[159]. The authors reported that vaso-spasm induced ischemia is the cause for short-lasting vertigo attacks as

part of the aura and this might lead to irreversible labyrinth damage in migraine patients[160].

In addition, findings in migraine patients with vertigo might be based on different pathomechanisms like recurrent transient ischemias, serotoninergic induced extravasation causing peripheral vestibular deficits leading to labyrinthine signs and symptoms.

The current data in our study of migraine patients without vertigo presenting with reduced VEMP amplitudes but normal latencies point to hypo-perfusion induced ischemia or serotoninergic induced extravasation of the inner ear affecting the saccule rather than the brainstem.

On the other hand findings in migraine patients with vertigo presenting with more reduction of VEMP amplitude bilaterally signifies involvement of lesions in central vestibular processing. Considering that the assessment of the VEMPs was performed during the symptom free interval and the wide variation between the last migraine attack and the examination, the present results of reduced VEMP amplitudes during the interictal period makes it interesting. Thus, it seems that migraine attacks lead not only to transient effects but also to permanent deficits.

Although MV is considered to be very uncommon and atypicalpresentation of migraine , yet it is important to recognize this syndrome. Since treatments are available in relieving all or most of the symptoms, even in subjects who are unwell for years.

7. CONCLUSION

7. CONCLUSION:

VEMP a relatively new diagnostic test to assess the vestibular pathway was studied in relation to age , neck torsion and placement of inverting electrode. A sincere attempt was also made to study the changes in VEMP in migraine patients with age-sex matched controls.

VEMP response did not show much significant change in p13 and n23 latencies over different age groups but p13-n23 amplitude showed a significant decrease in the elderly which could be due to physiological degenerative changes of saccule and other vestibular apparatus including the vestibule-spinal tract due to advancing age.

Different modes of neck torsion did not produce any significant change in p13 or n23 latencies but there was a significant increase in p13-n23 amplitude when the head was lifted upwards and neck turned away from the source of stimulus indicating that the magnitude of VEMP amplitude correlates with the degree of neck torsion.

The more nearer the inverting electrode is to the active electrode greater is the VEMP response as evidenced by the robust amplitude seen when it was placed over the sternum.

The amplitude of VEMP in migraine patients, more so in migraine patients with vertigo was significantly lower compared to the controls probably due to the habituation deficit in cortical evoked potential which

might be associated with hypofunction of subcortical serotoninergic input to vestibular nucleus.

Although an honest attempt has been made, the study has its limitation since not much work has been done in the Indian sub-continent to compare the datas . Apart from inadequate number of subjects in the older age group a high level of motivation is necessary to elicit a good and robust response . A better understanding of the pathophysiology of migraine would throw more light on the usefulness of VEMP to demarcate migraine and vertigo associated with migraine. Further studies is being done on the usefulness of VEMP on this line.

8. SUMMARY

8. SUMMARY

VEMP arelatively new diagnostic tool was studied on 50 migraine patients and compared with age and sex matched controls. There was no significant variations of p13 and n23 latencies in either the controls or the migraine patients. The latencies did not change with regard to the age , different modes of neck torsion or the placement of inverting electrodes at different sites. P13-n23 ampltude was significantly higher when the neck was lifted forwards and turned away from the source of stimulus. Similarly the amplitude was greater when the reference electrode was placed at the sternum compared to wrist and mastoid. The amplitude decreases significantly after 50 years of age. In migraine patients with or without vertigo the amplitude showed a significant decrease .the magnitude of decrease in migraine patients was much higher.

9. BIBLIOGRAPHY

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10. ANNEXURES

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. S. Anbuselvi Mattuvar Kuzhali
PG in MD Physiology
Madras Medical College, Chennai -3

Dear Dr. S. Anbuselvi Mattuvar Kuzhali

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled " Vestibular Evoked Myogenic Potential in Migraine and healthy subjects " No:09032012.

The following members of Ethics Committee were present in the meeting held on 22.03.2012 conducted at Madras Medical College, Chennai -3.

1.	Prof. S.K. Rajan. MD	-- Chairperson
2	Prof. Pregna B. Dolia MD Vice Principal, Madras Medical College, Chennai -3 (Director , Institute of Biochemistry, MMC, Ch-3)	-- Member Secretary
3.	Prof. B. Kalaiselvi. MD Prof of Pharmacology ,MMC, Ch-3	-- Member
4.	Prof. C. Rajendiran, MD Director , Inst. Of Internal Medicine, MMC, Ch-3	-- Member
5.	Thiru. S. Govindsamy. BA BL	-- Lawyer
6.	Tmt. Arnold Soulina MA MSW	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

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INFORMED CONSENT FORM

(This is only a guideline – Relevant changes to be made as per the study requirements)

Title of the study:"_____".

Name of the Participant: _____.

Name of the Principal (Co-Investigator): _____.

Name of the Institution: _____.

Name and address of the sponsor / agency (ies) (if any):_____
_____.

Documentation of the informed consent

I _____ have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered. I am over 18 years of age and, exercising my free power of choice, hereby give my consent to be included as a participant in "_____

_____" (title of the study).

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.
3. I have been explained about the nature of the study.
4. I have been explained about my rights and responsibilities by the investigator.
5. I have been informed the investigator of all the treatments I am taking or have taken in the past _____ months including any native (alternative) treatment.
6. I have been advised about the risks associated with my participation in this study.*
7. I agree to cooperate with the investigator and I will inform him/her immediately if I suffer unusual symptoms. *
8. I have not participated in any research study within the past _____month(s). *
9. I have not donated blood within the past _____ months—Add if the study involves extensive blood sampling. *
10. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital. *
11. I am also aware that the investigator may terminate my participation in the study at any time, for any reason, without my consent. *
12. I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, Govt. agencies, and IEC. I understand that they are publicly presented.
13. I have understand that my identity will be kept confidential if my data are publicly presented
14. I have had my questions answered to my satisfaction.
15. I have decided to be in the research study.

I am aware that if I have any question during this study, I should contact the investigator. By signing this consent form I attest that the information given in this document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

For adult participants:

Name and signature / thumb impression of the participant (or legal representative if participant incompetent)

Name _____ Signature_____ Date_____

Name and Signature of impartial witness (required for illiterate patients):

Name _____ Signature_____ Date_____

Address and contact number of the impartial witness:

Name and Signature of the investigator or his representative obtaining consent:

Name _____ Signature_____ Date_____

For Children being enrolled in research:

Whether child's assent was asked: Yes / No (Tick one)
[If the answer to be above question is yes, write the following phrase:
You agree with the manner in which assent was asked for from your child and given by your child. You agree to have your child take part in this study].
[If answer to be above question No, give reason (s) :_____.

Although your child did not or could not give his or her assent, you agree to your child's participation in this study.

Name and Signature of / thumb impression of the participant's parent(s) (or legal representative)
Name _____ Signature_____ Date_____
Name _____ Signature_____ Date_____

Name and Signature of impartial witness (required for parents of participant child illiterate):

Name _____ Signature_____ Date_____

Address and contact number of the impartial witness: _____

Name and Signature of the investigator or his representative obtaining consent :

Name _____ Signature_____ Date_____

NOTE-

For observational studied in nature or those in which only patient's tissue, body fluids are collected for any kind of analysis, the following elements in the patient information leaflet will need be included – background of the study: the purpose for which the sample will be used: confidentially of data are right to refuse to give specimens should be included.

Points 6, 7,8,9,10,11 of consent document may be excluded in such cases.

PROFORMA

ASSESSMENT OF VESTIBULAR EVOKED MYOGENIC POTENTIAL

SERIAL NO: _____ DATE: _____

Name: Age: Sex: M / F

MIGRAINE: Age of onset:

Temporal relationship:

a) Migraine first
b) Vertigo first
c) Together

Frequency of the attacks:

a) Once or twice/month
b) Once or twice/week
c) Daily
d) Occasional

Severeity:

Grade 1 : patient still able to perform normal work activity

Grade 2: unable to perform normal work but bed rest is unnecessary

Grade 3: bed rest is necessary.

VERTIGO :

Frequency of the attacks:

a) Once or twice/month
b) Once or twice/week
c) Daily
d) Occasional

Severeity :

Grade 1 : patient still able to perform normal work activity

Grade 2: unable to perform normal work but bed rest is unnecessary

Grade 3: bed rest is necessary.

DURATION :

Migrainous attack - Short. Vertigo attack - Hours

Long Minutes

SITE : Bifrontal Right Frontal Left Frontal

VESTIBULAR SYMPTOMS :

i. Head Motion Intolerance
ii. Positional Vertigo
iii. Rotational Vertigo

PRESENCE OF AURA:

With Aura Without Aura

MIGRAINOUS SYMPTOMS DURING VERTIGINOUS ATTACKS

a) Headache
b) Phonophobia
c) Photophobia
d) Nausea
e) Vomiting
f) Visual & other auras

FAMILY H/O MIGRAINE :

H/O DRUG INTAKE :

H/O DM/HT :

H/O SMOKING : Yes / No. H/O ALCOHOL : yes/no.

AUDIOLOGICAL EXAMINATION :

AUDIOMETRY :

EXAMINATION OF CVS :

EXAMINATION OF RS :

EXAMINATION OF CNS :

Motor system

Sensory system

Co-ordination

Cranial nerve examination

MIGRAINE WITH VERTIGO								**B/L LEFT**		**B/L RIGHT**			**U/L RT HR**			**U/L LT HR**					
name	gender	AGE	HEIGHT	WEIGHT	BMI	P13	N23	P13-N23	P13	N23	P13-N23	P13	N23	P13-N23				IAR % B/L		IAR % U/L	
darmendran	male	17	165	67	24.6	12.97	22.35	27.89	12.29	24.53	26.06	11.51	22.81	27.22	14.9	23.44	28.68	-0.03392	-3.39203	-0.02612	-2.61181
AASHISH	male	17	163	81	30.5	11.25	19.48	38.2	11.56	21.98	32.32	15.37	22.45	45.55	10.9	17.45	38.04	-0.08338	-8.33806	0.089843	8.984328
bhanu	female	18	170	69	23.9	10.73	20.21	36.66	11.36	16	32.19	12.92	23.54	36.18	11.4	22.92	32.75	-0.06492	-6.49237	0.049761	4.976063
rajalakshmi	female	20	167	65	23.3	16.98	22.4	19.77	14.43	22.5	21.2	13.6	18.28	19.57	14.7	21.15	23.3	0.034904	3.490359	-0.08701	-8.70072
madina begum	female	21	165	70	25.7	11.98	19.85	29.4	11.41	22.87	23.84	0.73	23.75	16.93	12.5	20.31	25.17	-0.10443	-10.4433	-0.19572	-19.5724
karthikeyan	male	22	162	85	32.4	12.61	24.17	18.38	15	18.8	26.06	12.5	23.44	24.02	12	24.38	25.19	0.172817	17.28173	-0.02378	-2.37757
kawtham	male	23	174	84	27.7	12.08	23.02	20	15.58	22.5	19.67	11.56	20.42	27.7	13.7	21.56	23.07	-0.00832	-0.83186	0.091196	9.119559
SUKANYA	female	24	155	69	28.7	10.42	25.1	28.88	13.86	21.25	26.89	13.66	19.89	28.45	13.3	22.66	27.13	-0.03568	-3.56823	0.02375	2.374955
parameshwary	female	25	169	63	22.1	11.98	23.65	26.13	12.19	23.6	32.89	10.79	20.42	33.33	12.3	24.22	33.48	0.114537	11.45374	-0.00225	-0.22452
tamilarasi	female	26	170	72	24.9	13.18	23.7	31.12	10.42	21.69	30.16	15.63	23.23	28.5	13.2	18.44	28.53	-0.01567	-1.56658	-0.00053	-0.0526
susheela	female	30	168	52	18.4	9.48	20.4	22.15	10.83	21.78	18.53	10.83	21.78	18.16	10.9	18.54	24.22	-0.08899	-8.89872	-0.14299	-14.2992
ramesh	male	31	161	70	27	12.19	21.3	26.27	12.19	22.08	30.62	12.19	21.67	30.93	14.2	21.67	24.69	0.076463	7.646335	0.11219	11.21899
meena	female	32	160	63	24.6	11.41	17.71	23.89	10.42	21.57	17.65	10.42	21.77	18.14	11.5	24.38	19.08	-0.15022	-15.0217	-0.02526	-2.52552
Thiyagarajan	male	35	172	82	27.7	10.63	17.4	21.62	11.98	20.42	17.17	10.68	15.47	11.93	10.7	24.27	17.39	-0.11472	-11.472	-0.18622	-18.6221
kalyanasundaram	male	36	157	42	17	10.73	24.69	26.35	11.84	23.04	23.52	13.23	21.46	20.13	11.8	23.85	16.13	-0.05675	-5.67475	0.110314	11.03144
hemalatha	female	37	170	76	26.3	11.15	23.3	22.78	10.78	27.97	25.68	10.83	27.5	24.04	10.9	24.9	25.42	0.059843	5.984317	-0.0279	-2.79013
amsa	female	39	155	65	27.1	10.63	18.44	27.4	13.54	19.58	27.6	11.36	22.92	24.23	10.4	26.25	27.11	0.003636	0.363636	-0.0561	-5.60966
lakshminarayanan	male	40	167	78	28	10.89	17.5	24.64	10.63	18.13	23.44	10.83	17.97	25.52	10.6	0.08	24.27	-0.02496	-2.49584	0.025105	2.510544
rajam	female	41	161	67	25.8	10.73	23.02	25.57	11.25	18.44	23.79	11.25	18.44	25.25	10.9	18.44	22.03	-0.03606	-3.60616	0.068105	6.810491
poongavanam	female	42	165	70	25.7	11.15	22.19	14.65	12.66	21.88	18.46	12.5	21.98	18.21	10.4	24.27	19.22	0.115071	11.5071	-0.02698	-2.69837
selvi	female	45	155	53	22.1	11.15	24.33	11.13	10.42	21.98	14.25	12.5	21.98	10.98	10.8	24.27	15.48	0.122931	12.29314	-0.17007	-17.0068
latheefa	female	46	164	80	29.7	13.39	21.98	19.59	11.56	21.42	23.86	10.73	25	28.63	13	22.97	25.34	0.098274	9.827388	0.06096	6.095979
murugan	male	47	165	60	22	13.13	20.1	32.36	10.94	20	38.88	11.98	24.12	32.54	11.3	25.47	32.8	0.091522	9.152162	-0.00398	-0.39792
mary m	female	48	168	71	25.2	12.35	21.67	12.46	10.42	22.71	13.15	10.42	23.13	16.83	11.7	22.35	13.62	0.026943	2.69426	0.105419	10.54187
amudha	female	50	171	73	25	10.42	22.5	18.57	12.19	26.5	16.57	12.19	22.61	18.66	10.4	22.5	15.84	-0.05692	-5.69152	0.081739	8.173913
mahalakshmi N	female	52	170	74	25.6	10.42	27.55	15.16	10.42	27.08	15.88	12.71	22.12	14.51	11.7	24.17	15.81	0.023196	2.319588	-0.04288	-4.2876
suganthi	female	53	168	65	23	11.88	22.71	16.93	10.99	22.45	11.49	10.99	22.4	12	11.9	23.33	12.78	-0.19141	-19.1414	-0.03148	-3.1477
tamilselvi	female	55	170	62	29.5	17.09	23.08	6.81	12.3	21.34	8.29	14.1	24.27	6.94	10.1	23.06	6.3	0.098013	9.801325	0.048338	4.833837
PRAKASH M	male	61	160	70	27.3	12.11	20.63	12.49	12.23	18.75	15.34	12.24	18.75	17.27	12.5	20.64	14.49	0.102407	10.24075	0.087531	8.753149
KALADEVI	female	63	166	65	23.6	12.67	18.88	17.78	12.04	19.17	18.27	12.71	19.17	18.03	12.9	18.87	17.87	0.013592	1.359223	0.004457	0.445682

MIGRAINE WITHOUT VERTIGO

						B/L LEFT			**B/L RIGHT**			**U/L RT HR**			**U/L LT HR**					
name	gender	AGE	HEIGHT	WEIGHT	BMI	P13	N23	P13-N23	P13	N23	P13-N23	P13	N23	P13-N23	P13	N23	P13-N23	IAR % B/L		IAR % U/L	
indhu	female	17	166	68	24.7	11.36	17.4	27.45	10.42	20.29	28.72	13.13	21.57	28.05	10.42	26.25	22.42	0.02261	2.260993	0.111551	11.15514
shabeena	female	18	160	55	21.5	12.5	18.27	36.76	10.42	22.26	24.85	11.98	20.42	27.17	11.57	22.97	26.4	-0.19331	-19.3313	0.014374	1.437372
vigneshwari	female	21	170	74	25.6	11.77	21	37.65	11.04	21.35	48	13.91	21.6	39.6	10.58	21.58	32.37	0.120841	12.08406	0.100459	10.04585
gayathri	female	25	162	70	26.7	11.04	22.19	20.93	14.27	21.45	29.03	10.89	21.77	33.1	11.56	21.58	26.83	0.16213	16.21297	0.104622	10.46221
latha n	female	26	175	90	29.4	11.56	18.54	25.17	10.73	17.4	25.06	10.83	17.45	19.52	10.73	18.44	19.54	-0.00219	-0.21899	-0.00051	-0.0512
mohamedkadarnus	male	27	168	70	24.8	10.83	20.57	27.29	11.57	23.33	28.75	13.65	20.42	26.08	11.98	22.92	21.6	0.026053	2.605282	0.09396	9.395973
rajalakshmi	female	29	162	68	25.9	13.23	16.67	25.89	14.79	20.24	30.23	11.64	21.6	28.76	12.71	23.23	26.37	0.077334	7.733428	0.043352	4.335208
selvi	female	29	165	70	25.7	11.63	23.03	27.58	10.42	23.75	23.75	12.56	21.35	17.17	11.67	24.59	24.54	-0.07462	-7.46152	-0.1767	-17.6696
radha	female	30	173	69	23.1	13.96	24.9	37.17	13.23	21.2	15.06	10.63	23.62	22.63	11.15	24.69	21.11	-0.42332	-42.332	0.034751	3.47508
sngeetha	female	32	161	63	24.3	12.27	22.6	26.93	10.52	22.68	27.23	10.73	16.1	32.92	13.17	22.6	35.59	0.005539	0.553914	-0.03897	-3.89724
mutharasan	male	33	167	73	26.2	11.46	17.24	27.87	11.04	21.04	30.48	10.83	22.19	28.01	10.94	18.44	26.73	0.04473	4.473008	0.023383	2.338327
malliga	female	37	170	65	22.5	10.42	22.6	8.74	12.19	22.4	9.32	12.19	22.4	10.08	10.42	22.5	8.75	0.032115	3.211517	0.070632	7.063197
uma	female	37	165	55	20.2	12.17	24.8	25.46	15.31	22.82	25.9	11.88	21.25	22.51	13.28	25.06	21.11	0.008567	0.856698	0.032095	3.209537
mahalakshmi	female	40	174	70	23.1	11.22	22.19	19.58	12.19	23.2	20.81	12.48	20.33	19.78	12.42	15.21	23.93	0.030453	3.045308	-0.09494	-9.49439
mary	female	42	160	65	25.4	12.6	23.33	13.66	12.92	23.33	18.78	13.54	21.88	24.7	10.63	26.04	20.81	0.15783	15.78298	0.085476	8.547572
s shanthi	female	46	168	66	23.4	10.94	17.14	30.37	10.73	22.35	23.13	10.73	16.41	24.43	10.94	18.44	21.38	-0.13533	-13.5327	0.066579	6.657935
kumari	male	47	163	66	24.8	10.52	22.5	21.1	11.2	21.93	21.73	12.19	21.98	16.89	12.42	22.5	23.33	0.014709	1.470932	-0.16012	-16.0119
neelaveni	female	50	174	71	23.5	11.06	21.77	17.29	10.63	23.54	25.66	11.51	22.82	29.18	11.72	22.08	21.7	0.194878	19.48778	0.147013	14.70126
chandra	female	52	166	71	25.8	10.94	19.58	11.27	10.83	19.6	10.74	10.83	19.6	10.73	10.94	19.74	11.35	-0.02408	-2.408	-0.02808	-2.80797
sangeetha .P	female	55	175	80	26.1	13.08	20.13	18.81	12.04	20.1	13.42	12.63	23.03	11.66	14.59	20.11	10.23	-0.16724	-16.7235	0.065327	6.532663

80 CONTROL GROUP					B/L LEFT			B/L RIGHT			U/L RT HR			U/L LT HR							
	gender	AGE	HEIGHT	WEIGHT	P13	N23	P13-N23	P13	N23	P13-N23	P13	N23	P13-N23	P13	N23	P13-N23	P13	N23	P13-N23	B/L	U/L	
NANDINI	female	17	170	54	11.04	18.44	52.54	16.36	21.57	46.78	9.74	19.95	60.49	9.69	16.35	59.69	11.7075	19.0775	54.875	-0.05799	-5.79944	0.006657	0.665668
SANGEETHA	female	17	160	60	10.63	21.92	40.93	10.63	21.44	39.03	10.42	22.14	56.25	11.04	21.78	54.28	10.68	21.82	47.6225	-0.02376	-2.37619	0.017823	1.782322
HARIHARAN	male	17	170	47	10.42	18.44	52.14	10.47	16.62	59.01	10.42	17.4	50.23	10.42	18.44	51.31	10.4325	17.725	53.1725	0.061808	6.180837	-0.01064	-1.06362
ABDUL RAJAK	male	17	174	61	11.72	24.27	56	10.42	18.44	58.23	10.63	18.44	57.25	10.63	14.54	56.98	10.85	18.9225	57.115	0.019522	1.952202	0.002364	0.236365
ABHSHEK	male	17	165	55	10.42	26.04	28.73	10.53	25.97	34.24	11.98	25.57	29.78	11.36	25.75	36.33	11.0725	25.8325	32.27	0.087502	8.750199	-0.09908	-9.90773
VAISHNAVI	female	18	172	50	10.42	17.87	49.78	14.17	21.83	45.8	12.97	20.83	47.09	10.73	18.44	48.93	12.0725	19.7425	47.9	-0.04164	-4.16405	-0.01916	-1.91627
RAJESH	male	18	171	55	16.94	19.17	36.19	13.13	21.72	41.29	13.44	22.29	39.16	12.45	23.39	36.83	13.99	21.6425	38.3675	0.065823	6.582344	0.030662	3.066193
SATHYASAGAR	male	18	168	60	13.23	21.98	41.15	11.7	18.75	42.18	12.5	18.54	42.1	12.5	21.53	42.23	12.4825	20.2	41.915	0.01236	1.236049	-0.00154	-0.15416
VISHNU	female	19	168	50	11.67	26.46	35.01	11.25	21.56	35.68	13.33	20.94	35.38	15.63	25.26	34.62	12.97	23.555	35.1725	0.009478	0.9478	0.010857	1.085714
VINODHINI	female	19	168	50	12.08	22.81	39.55	10.42	18.44	41.9	10.42	23.23	35.3	10.52	24.7	39.4	10.86	22.295	39.0375	0.028852	2.885206	-0.05489	-5.48862
VIJISHA	female	20	168	64	10.42	27.55	37.6	11.46	24.06	32.77	13.7	26.04	39.79	13.85	26.31	35.22	12.3575	25.99	36.345	-0.06864	-6.86372	0.060925	6.092521
SASHIKUMAR	male	20	168	67	10.52	25.21	45.16	11.2	24.17	43.16	10.42	24.17	48.1	10.42	25.83	46.95	10.64	24.845	45.8425	-0.02264	-2.26449	0.012099	1.20989
JAYACHANDRAN	male	21	170	65	12.5	18.44	47.79	12.67	20	53.66	10.94	19.48	42.48	10.73	19.38	43.94	11.71	19.325	46.9675	0.057861	5.786102	-0.01689	-1.68942
MEENAKUMARI	female	21	158	60	12.71	26.04	40.8	12.71	22.04	42.8	15.63	22.5	38.38	13.44	20.42	35.78	13.6225	22.75	39.44	0.023923	2.392344	0.035059	3.505933
BENETTA	female	22	160	60	17.5	21.88	38.7	12.82	25.83	42.83	10.83	23.64	39.18	10.53	21.67	41.62	12.92	23.255	40.5825	0.050656	5.06562	-0.0302	-3.0198
JAYASIMMAN	male	22	147	87	13.54	27.08	34.43	10.42	27.5	39.11	11.04	26.57	38.55	11.04	26.67	37.04	11.51	26.955	37.2825	0.063639	6.363884	0.019976	1.997619
KRISHNA	male	23	173	68	10.34	19.79	47.43	10.54	18.36	48.18	10.73	19.12	35.08	10.63	18.75	37.12	10.56	19.005	41.9525	0.007844	0.784437	-0.02825	-2.82548
SEERANGAMMAL	female	23	154	45	12.97	20.27	56.95	10.42	24.06	47.7	10.42	23.98	46.56	10.68	25.01	44.32	11.1225	23.33	48.8825	-0.08839	-8.83899	0.024648	2.464789
KASI RAJAN	male	24	160	60	13.85	19.17	55.67	12.19	18.44	59.6	11.26	18.83	45.01	11.67	19.56	42.19	12.2425	19	50.6175	0.034094	3.409387	0.032339	3.233945
POOVIZHI	female	24	165	55	10.73	14.64	50.64	10.53	24.48	42.8	14.12	24.38	48.7	12.14	24.58	45.73	11.88	22.02	46.9675	-0.0839	-8.39041	0.031452	3.145187
VANITHA	female	25	153	54	10.47	27.45	48	14.38	24.63	42.05	12.67	18.99	48	11.52	24.21	45.98	12.26	23.82	46.0075	-0.06607	-6.60744	0.021494	2.149393
LATHA	female	25	168	61	11.7	22.5	38.98	11.56	21.98	36.92	11.45	22.04	38.6	11.05	21.01	36.56	11.44	21.8825	37.765	-0.02714	-2.7141	0.027142	2.71421
PREETI	female	26	157	65	11.96	22.4	42.65	11.77	18.44	48.03	11.77	18.44	47.29	13.96	22.29	45.47	12.365	20.3925	45.86	0.05933	5.932951	0.019621	1.962053
RAJI	female	26	163	62	11.17	22.6	56.1	10.42	23.55	57.88	10.42	23.44	55.65	13.96	22.29	54.47	11.4925	22.97	56.025	0.015617	1.561677	0.010716	1.071558
KISHORE	male	27	158	66	11.08	18.44	48.66	10.83	18.44	48.09	10.83	18.44	49.68	12.08	18.55	49.34	11.205	18.4675	48.9425	-0.00589	-0.58915	0.003434	0.343365
KAUTHAM	male	28	154	65	11.19	18.44	67.44	10.83	25.63	54.08	10.83	25.63	51.34	12.29	18.44	45.44	11.285	22.035	54.575	-0.10994	-10.9941	0.060963	6.096301
FEBIN JOSEPH	male	29	150	55	11.87	24.06	52.26	10.63	25.21	49.78	10.63	26.51	46.68	13.96	23.85	43.38	11.7725	24.9075	48.025	-0.0243	-2.43042	0.036642	3.664224
ANBU	female	29	161	67	10.46	19.38	51.09	10.79	21.98	45.24	11.04	20.42	45.22	10.42	19.67	47.7	10.6775	20.3625	47.3125	-0.06073	-6.07287	-0.02669	-2.66896
CHANDRAKALA	female	30	156	60	10.42	18.44	45.14	13.86	21.25	46.89	13.45	20.18	46.6	13.66	19.89	48.45	12.8475	19.94	46.77	0.019016	1.901554	-0.01946	-1.94634
USHA	female	30	161	60	10.23	21.98	41.08	10.7	18.75	42.18	10.5	18.54	42.1	10.76	18.88	41.75	10.5475	19.5375	41.7775	0.013212	1.321163	0.004174	0.417412
RANDHEL SINGH	male	31	159	65	11.67	23.46	38.06	11.25	21.56	25.68	13.33	20.94	38.08	13.28	23.45	41.72	12.3825	22.3525	35.885	-0.19423	-19.4227	-0.04561	-4.5614
ISHA SOOD	female	32	149	60	11.01	21.81	42.33	12.92	17.14	44.8	12.5	16.88	43.6	12.67	18.44	44.23	12.275	18.5675	43.74	0.028348	2.834844	-0.00717	-0.71729
RATHI CHAWAN	female	32	157	69	10.83	23.72	47.63	10.75	23.54	42.72	10.98	22.94	39.18	11.71	23.72	42.67	11.0675	23.48	43.05	-0.05434	-5.43442	-0.04264	-4.2639
SASHI	male	33	158	72	12.81	18.75	43.12	11	21.77	47.43	13.7	21.77	49.4	12.81	22.08	45.28	12.58	21.0925	46.3075	0.047598	4.759801	0.043515	4.3515
VISHWANATH	male	34	163	73	13.54	22.08	51.88	11.25	27.29	52.35	12.5	24.48	51.94	12.6	23.02	51.16	12.4725	24.2175	51.8325	0.004509	0.450926	0.007565	0.756547
SATHYANARAYANAN	male	34	168	90	11.15	19.01	43.23	10.52	18.96	43.86	10.42	21.98	43.33	10.42	24.33	40.08	10.6275	21.07	42.625	0.007234	0.72339	0.038964	3.896415
ARUL PRASANNA	male	35	173	75	11.67	21.26	33.48	10.52	20.42	26.29	11.67	21.72	39.97	11.67	21.88	39.76	11.3825	21.32	34.875	-0.12029	-12.0294	0.002634	0.263389
RAJARAMAN	male	36	170	68	13.96	18.39	37.92	12.81	18.86	35.35	13.7	19.79	35.73	13.44	19.79	40.22	13.4775	19.2075	37.305	-0.03508	-3.50757	-0.05912	-5.91178
VASUDEVAN	male	36	154	55	10.63	19.58	38.67	10.78	19.38	37.2	10.78	21.55	40.72	11.57	21.76	38.79	10.94	20.5675	38.845	-0.01938	-1.93752	0.024274	2.427368
GIRIJA	female	37	162	62	10.42	18.44	42.98	10.83	26.15	41.52	12.6	20.52	43.2	10.63	23.44	43	11.12	22.1375	42.675	-0.01728	-1.72781	0.00232	0.232019
PRAKASH	male	38	169	67	11.67	25.83	36.89	11.73	25.56	34.63	11.73	25.56	39.47	11.67	25.83	38.69	11.7	25.695	37.42	-0.0316	-3.15996	0.00998	0.997953
RAMESH KRISHNAN	male	38	170	83	11.77	21.76	39.47	11.27	22.88	38.91	11.72	22.38	33.57	11.56	21.86	32.96	11.58	22.22	36.2275	-0.00714	-0.71447	0.009169	0.91688
KAVITHA	female	39	152	72	11.35	19.27	45.95	14.17	21.67	45.24	12.5	20.42	48.28	11.35	20.42	46.6	12.3425	20.445	46.5175	-0.00779	-0.77859	0.017707	1.770658
ELUMALAI	male	40	155	57	10.42	24.27	41.19	10.52	18.96	48.49	10.42	24.33	40.79	10.58	24.27	41.31	10.485	22.9575	42.945	0.081401	8.140054	-0.00633	-0.63337

RAVI P	female	40	157	49	10.63	17.24	39.13	11.38	24.63	40.05	11.67	18.99	38	11.52	24.21	32.98	11.3	21.2675	37.54	0.011619	1.16191	0.070724	7.072415
PREETHA	female	41	175	65	11.98	22.44	36.14	11.3	24.17	38.12	12.29	24.17	47.59	12.29	22.92	41.09	11.965	23.425	40.735	0.026663	2.666308	0.073297	7.329725
MOHAN	male	41	151	62	12.5	23.33	38.89	13.02	23.13	38.95	12.08	21.67	38.09	12.5	23.33	38.99	12.525	22.865	38.73	0.000771	0.077081	-0.01168	-1.16762
S SARASWATHY	female	42	154	49	12.31	22.2	42.27	12.5	21.35	41.52	11.48	22.87	44.8	11.52	25.32	38.53	11.9525	22.935	41.78	-0.00895	-0.89509	0.075243	7.524301
SASIKUMAR	male	43	158	52	10.52	22.21	42.98	10.38	22.63	46.05	10.44	23.58	44.92	10.42	21.67	41.12	10.44	22.5225	43.7675	0.034483	3.448276	0.044166	4.41655
SIBI	male	44	166	59	13.96	22.4	40.58	11.77	18.44	43.78	14.96	22.4	42.85	13.96	18.44	43.74	13.6625	20.42	42.7375	0.037933	3.793267	-0.01028	-1.02783
KARTHICK	male	45	162	69	10.42	21.98	32.19	10.42	24.58	36.09	10.42	26.15	35.35	10.42	22.19	42.84	10.42	23.725	36.6175	0.057118	5.711775	-0.09579	-9.57923
AKALYA	female	45	161	55	12.71	23.04	39.09	12.71	23.94	40.16	12.71	23.02	38.94	12.51	22.04	37.6	12.66	23.01	38.9475	0.013502	1.350158	0.017507	1.750719
AMBIGA	female	46	177	65	12.6	19.27	44.29	10.13	19.27	49.88	10.23	22.01	52.59	12.78	19.7	46.56	11.435	20.0625	48.33	0.059361	5.936073	0.060817	6.081694
VARUN	male	47	181	60	11.18	20.81	45.44	12.67	22.01	41.2	12.18	21.81	43.81	11.67	22.01	46.3	11.925	21.66	44.1875	-0.04894	-4.89381	-0.02763	-2.76329
BALAJI	male	47	150	58	10.83	21.88	35.75	10.92	22.08	36.79	10.92	21.35	38.88	10.83	21.78	34.39	10.875	21.7725	36.4525	0.014337	1.433692	0.06128	6.12802
ANISHU	female	48	152	54	10.55	18.75	47.89	10.48	18.86	37.97	10.54	18.96	38.49	10.44	18.85	38.66	10.5025	18.855	40.7525	-0.11554	-11.5537	-0.0022	-0.22035
ANITHA	female	50	158	60	12.71	24.04	35.7	10.53	24.48	37.8	10.47	25.99	35.12	13.03	24.06	32.37	11.685	24.6425	35.2475	0.028571	2.857143	0.040747	4.074678
CHANDRU	male	50	174	72	11.35	19.27	32.98	12.19	18.54	39.86	12.19	18.54	35.47	12.21	18.44	38.61	11.985	18.6975	36.73	0.094454	9.44536	-0.04239	-4.23866
DIVYA	female	51	169	64	12.29	18.45	28.88	12.04	24.17	32.89	12.29	18.45	33.4	11.56	24.17	30.62	12.045	21.31	**31.4475**	0.064918	6.491825	0.043424	4.342393
KANCHANA	female	52	153	55	12.63	21.98	33.59	11.52	22.04	35.31	11.04	21.98	35.87	12.18	22.04	32.49	11.8425	22.01	**34.315**	0.024964	2.496372	0.049444	4.944412
EESHWARI	female	52	158	67	13.38	22.92	31.7	13.58	22.81	36.47	13.52	24.58	40.13	13.45	21.67	32.91	13.4825	22.995	**35.3025**	0.069972	6.997213	0.09885	9.884995
GOMATHY	female	53	161	56	10.42	23.7	31.69	10.52	23.29	24.32	10.42	23.5	27.85	10.42	21.77	31.65	10.445	23.065	**28.8775**	-0.13158	-13.1584	-0.06387	-6.38655
GEETHUKISHAN	female	54	163	50	10.63	18.75	27.54	10.63	18.75	26.7	10.83	19.06	25.08	10.73	18.75	30.04	10.705	18.8275	**27.34**	-0.01549	-1.54867	-0.08999	-8.99855
SUMAN	male	55	170	66	12.5	20.08	32.43	12.43	21.44	29.74	12.02	19.88	39.01	12.5	19.98	38.89	12.3625	20.345	**35.0175**	-0.04327	-4.32685	0.00154	0.154044
ANJU	female	55	159	51	11.04	21.67	26.57	11.04	22.65	34.19	11.04	21.65	28.24	11.04	21.76	28.88	11.04	21.9325	**29.47**	0.125411	12.54115	-0.0112	-1.12045
RAO	male	57	183	70	12.81	22.6	22.18	11.56	21.98	26.92	11.45	22.04	29.6	11.45	21.01	28.56	11.8175	21.9075	**26.815**	0.096538	9.653768	0.017882	1.788171
BHARATH	male	58	155	52	11.71	21.24	36.28	11.65	21.42	37.22	11.62	22.34	35.73	11.76	22.45	28.49	11.685	21.8625	**34.43**	0.012789	1.278912	0.112737	11.27375
SURESH BABU	male	60	157	69	10.55	18.75	17.89	10.48	18.86	17.97	10.54	18.96	18.49	10.44	18.85	18.66	10.5025	18.855	**18.2525**	0.002231	0.22309	-0.00458	-0.4576
ZAHIR	male	61	158	63	10.6	23.02	17.57	11.6	22.08	17.88	11.56	22.08	16.67	11.67	23.02	14.83	11.3575	22.55	16.7375	0.008745	0.874471	0.058413	5.84127
PRAKASH M	male	61	160	65	12.11	20.63	22.49	12.23	18.75	25.34	12.24	18.75	27.27	12.48	20.64	24.49	12.265	19.6925	24.8975	0.059586	5.958603	0.053709	5.370943
MICHAEL	male	61	171	57	12.11	21.07	18.05	11.56	21.07	17.59	11.55	21.07	20.69	12.12	21.44	16.47	11.835	21.1625	18.2	-0.01291	-1.29068	0.113563	11.3563
DINESH	male	62	168	67	12.89	18.16	16.05	12.88	18.72	18.84	12.97	18.93	18.03	12.89	18.28	17.06	12.9075	18.5225	17.495	0.079966	7.996561	0.027643	2.76432
RAJAGOPAL	male	63	152	54	10.67	19.27	18.32	10.56	21.67	25.71	11.66	21.56	23.56	11.57	21.4	18.43	11.115	20.975	21.505	0.16784	16.78401	0.122172	12.21719
SUMATHI	female	63	149	60	10.52	20.21	17.98	10.52	23.33	17.05	11.54	22.56	17.92	11.52	22.5	21.73	11.025	22.15	18.67	-0.02655	-2.65487	-0.09609	-9.60908
RAJESH	male	64	152	55	10.69	22.89	22.47	10.76	22.87	25.87	10.59	22.98	25.59	10.78	22.98	21.88	10.705	22.93	23.9525	0.070335	7.033513	0.078155	7.815462
RATHINAVEL	male	65	153	78	13.07	18.44	17.2	13.32	18.56	18.82	13.17	18.56	19.43	13.13	18.44	16.93	13.1725	18.5	18.095	0.044975	4.497501	0.068757	6.875688
KUPPU	female	66	162	61	10.52	21.88	18.12	10.52	21.78	16.53	10.55	21.66	16.05	10.54	21.77	18.93	10.5325	21.7725	17.4075	-0.04589	-4.58874	-0.08233	-8.23328
PADMASINI	female	67	167	73	11.25	24.58	15.41	12.15	24.44	18.98	11.25	24.43	15.78	11.52	21.58	20.19	11.5425	23.7575	17.59	0.103809	10.38092	-0.1226	-12.2602
JULIA	female	68	166	74	11.43	21.56	19.9	11.43	21.88	19.91	11.43	21.88	18.29	11.43	21.56	17.83	11.43	21.72	18.9825	0.000251	0.025119	0.012735	1.273533
HEMAMBUJAVALLI	female	70	165	66	10.42	24.06	12.92	10.98	21.15	15.41	10.98	20.97	18.9	10.91	23.35	17.82	10.8225	22.3825	16.2625	0.087893	8.789269	0.029412	2.941176

20CONTROLS FOR REFERENCE				STERNUM				WRIST				MASTOID			
p13 latency				B/L LT	B/L RT	U/L RT	U/L LT	B/L LT	B/L RT	U/L RT	U/L LT	B/L LT	B/L RT	U/L RT	U/L LT
ARUL PRASSANNA	17	173	90	11.67	10.52	10.52	11.67	11.25	10.52	10.52	11.67	11.67	10.52	10.52	11.67
FEBIN JOSEPH	17	172	61	14.67	14.52	14.52	14.67	11.25	12.52	2.52	11.67	11.67	12.52	12.52	11.67
VIVEKANAND	17	171	55	11.67	10.52	10.52	11.67	11.25	10.52	10.52	11.67	11.67	10.52	10.52	11.67
SASHIREKA	18	168	61	12.81	10.99	13.7	12.81	12.81	10.75	13.75	12.81	12.81	10.75	13.75	12.81
VARSHINI	18	168	50	12.71	10.73	13.75	12.81	12.4	10.75	11.75	12.81	12.81	11.75	11.75	12.81
ANUP	18	168	64	12.81	11.25	13.65	12.81	12.81	11.75	11.75	12.81	12.81	11.65	11.62	12.81
VISHWAM	19	168	67	13.54	14.25	12.5	12.6	12.7	12.69	12.81	12.71	12.1	12.81	13.33	12.81
VANDANA OJHA	19	168	65	14.34	13.25	13.5	13.65	12.6	12.56	12.81	12.6	12.6	11.81	11.33	12.81
TARUN	19	170	60	13.54	11.25	12.5	12.6	12.8	12.81	12.81	12.81	12.81	11.61	11.33	12.81
RITESH	20	154	60	12.76	11.45	12.67	12.55	12.75	12.65	12.72	12.56	12.71	12.37	12.77	12.31
SOWMYA	20	160	87	11.93	11.78	11.92	11.78	12.28	12.01	11.98	11.78	11.01	11.56	11.67	11.56
SHANTHINI	21	159	68	12.41	11.69	11.73	12.41	11.72	11.08	11.18	11.72	11.62	11.08	11.18	11.62
PRATIMA	21	165	45	11.92	11.56	11.65	11.92	11.59	11.83	11.73	11.69	11.04	10.83	10.89	11.04
SELVI	22	153	60	13.85	15.63	15.43	12.08	11.56	13.23	12.4	13.23	12.45	11.84	13.85	11.56
SUGAPRIYA	23	168	70	12.22	12.42	12.89	13.32	10.42	11.08	11.9	10.62	11.61	10.43	10.56	11.67
GANDHIMATHI	23	174	55	13.01	13.75	13.45	13.44	11.25	11.15	12.97	12.79	11.67	11.56	11.72	12.04
VENKATASELVAM	24	165	54	13.24	13.75	13.33	13.54	13.67	11.23	11.76	11.03	13.45	13.28	13.02	13.19
DEVI	24	174	61	13.19	13.85	13.42	11.47	12.49	12.58	12.09	13.23	12.14	13.26	12.76	13.14
DHANAM	25	172	70	12.86	13.88	12.58	13.18	11.82	11.48	11.56	11.05	11.83	11.74	10.73	11.63
YALINI DEVI	25	171	55	12.59	12.61	12.98	13.06	11.44	11.57	11.67	11.54	11.82	11.81	10.73	11.64
KUMARAN DEV	26	168	64	14.42	14.79	14.06	14.16	11.33	11.69	12.48	11.89	12.83	12.63	12.67	12.98
SANTOSHVARAN	26	168	61	11.43	11.46	11.73	11.49	10.42	10.88	10.92	10.52	11.02	11.91	11.89	11.73

				STERNUM				WRIST				MASTOID			
n23 latency				B/L LT	B/L RT	U/L RT	U/L LT	B/L LT	B/L RT	U/L RT	U/L LT	B/L LT	B/L RT	U/L RT	U/L LT
ARUL PRASSANNA	17	173	90	21.26	20.42	18.44	21.88	24.17	18.54	18.44	24.17	22.82	18.44	18.44	24.54
FEBIN JOSEPH	17	172	61	20.63	20.42	18.44	21.88	24.17	18.54	18.44	24.17	21.46	18.44	18.44	24.9
VIVEKANAND	17	171	55	21.88	20.42	18.44	21.88	24.17	18.54	18.44	24.17	24.17	18.44	18.44	24.17
SASHIREKA	18	168	61	22.85	21.77	21.77	22.08	18.75	21.04	21.98	18.75	18.75	21.04	21.04	18.75
VARSHINI	18	168	50	22.75	21.77	21.77	22.08	18.75	21.04	21.98	18.75	18.75	21.04	21.04	18.75
ANUP	18	168	64	19.99	21.77	21.77	22.08	18.75	21.04	21.98	18.75	18.75	21.04	21.04	18.75
VISHWAM	19	168	67	22.08	27.29	27.19	23.02	20.73	21.77	21.77	18.75	23.02	27.19	27.19	23.02
VANDANA OJHA	19	168	65	22.08	27.29	21.77	23.02	18.44	21.77	21.77	18.75	23.02	27.19	21.77	23.02
TARUN	19	170	60	22.08	27.29	24.48	23.02	23.02	21.77	21.77	18.75	23.02	27.19	24.48	23.02
RITESH	20	154	60	23.12	23.43	23.08	23.12	22.67	22.66	22.72	22.81	22.98	22.67	22.96	22.78

SOWMYA	20	160	87	23.18	23.09	23.58	23.18	23.01	22.79	22.59	22.93	22.44	22.56	22.67	22.48
SHANTHINI	21	159	68	18.81	18.23	18.66	18.73	17.66	18.01	17.98	17.69	18.94	18.23	17.73	17.45
PRATIMA	21	165	45	19.83	19.97	19.63	19.55	19.03	8.17	19.17	20.61	19.73	19.07	18.99	18.98
SELVI	22	153	60	21.56	21.89	21.74	22.55	19.89	19.15	19.42	19.49	22.07	19.98	19.33	18.77
SUGAPRIYA	23	168	70	24.55	22.85	22.96	24.69	18.45	18.19	18.56	20.78	21.93	21.11	21.15	21.57
GANDHIMATHI	23	174	55	24.43	20.23	23.56	23.65	23.51	24.85	23.57	23.75	23.51	23.45	23.25	23.01
VENKATASELVAM	24	165	54	24.93	24.39	24.39	24.47	20.14	20.63	20.36	20.3	20.56	20.95	20.85	20.41
DEVI	24	174	61	24.08	23.32	23.23	24.11	22.53	22.25	23.15	22.74	22.69	22.96	23.48	23.84
DHANAM	25	172	70	20.94	20.81	20.93	21.33	20.33	20.47	20.73	20.84	21.63	21.89	21.47	21.03
YALINI DEVI	25	171	55	22.83	23.07	22.03	24.44	20.21	20.8	20.74	19.74	20.43	23.06	20.23	20.12
KUMARAN DEV	26	168	64	21.97	21.22	21.89	22.74	19.46	19.38	19.83	9.47	21.87	21.43	21.52	21.05
SANTOSHVARAN	26	168	61	24.67	24.06	23.76	24.76	22.56	22.22	23.22	21.56	23.66	23.87	24.03	22.88

				STERNUM					WRIST			MASTOID			
p13-n23				B/L LT	B/L RT	U/L RT	U/L LT	B/L LT	B/L RT	U/L RT	U/L LT	B/L LT	B/L RT	U/L RT	U/L LT
ARUL PRASSANNA	17	173	90	53.48	56.29	52.72	58.26	46.72	46	44.35	45.54	54.81	52.83	51.59	47.79
FEBIN JOSEPH	17	172	61	64.49	66.74	62.91	68.79	60.68	61.15	61.96	61.53	60.51	63.36	61.06	67.95
VIVEKANAND	17	171	55	52.47	52.84	53.52	52.73	46.75	45.84	43.74	45.67	51.1	51.29	48.12	47.62
SASHIREKA	18	168	61	58.05	57.84	58.03	55.71	48.76	47.11	48.31	49.08	51.27	51.39	51.14	51.77
VARSHINI	18	168	50	61.77	61.47	61.27	65.85	58.68	57.23	48.19	59	58.26	58.43	60.18	59.92
ANUP	18	168	64	57.87	58.2	57.78	55.57	55.83	56.98	55.43	55.15	51.28	53.34	52.09	55.61
VISHWAM	19	168	67	52.12	52.62	50.25	51.58	44.18	44.7	45.3	49.62	46.45	49.43	46.34	46.14
VANDANA OJHA	19	168	65	61.88	61.35	57.94	60.86	60.17	54.23	55.29	55.71	56.53	59.09	57.59	56.19
TARUN	19	170	60	51.73	52.08	55.63	51.45	47.18	45.17	45.31	49.52	50.36	49.76	45.08	46.09
RITESH	20	154	60	55.33	51.33	51.73	55.78	50.34	51.73	51.93	50.39	51.76	50.73	53.01	52.12
SOWMYA	20	160	87	56.85	57.74	57.76	57.38	55.65	54.83	50.73	52.13	53.34	54.76	53.17	53.84
SHANTHINI	21	159	68	60.53	59.74	59.38	56.63	53.24	53.72	53.81	53.42	55.44	55.76	55.76	55.44
PRATIMA	21	165	45	57.93	58.48	57.83	56.13	51.87	52.42	52.14	52.87	53.17	53.77	53.78	53.09
SELVI	22	153	60	51.34	51.83	59.32	51.87	50.93	50.87	50.22	51.93	50.67	50.38	51.86	51.57
SUGAPRIYA	23	168	70	54.77	54.56	52.01	48.77	50.01	51.2	51.2	50.07	48.56	48.73	48.38	48.92
GANDHIMATHI	23	174	55	55.67	55.71	55.23	55.01	50.74	50.39	50.82	50.19	52.18	52.12	52.23	52.43
VENKATASELVAM	24	165	54	54.47	49.98	49.98	53.47	47.77	45.01	45.01	47.77	51.22	47.59	46.59	50.45
DEVI	24	174	61	49.63	49.63	48.55	48.92	45.39	45.84	46.72	45.76	47.2	47.23	47.82	47.83
DHANAM	25	172	70	49.65	49.88	49.27	49.65	44.02	44.2	44.12	44.56	45.65	45.34	45.92	48.03
YALINI DEVI	25	171	55	53	51.17	52.38	53.54	52.74	52.98	52.88	52.96	51.18	51.24	52.38	52.37
KUMARAN DEV	26	168	64	53.33	51.83	51.27	51.28	51.57	51.83	51.24	52.74	50.55	50.57	50.84	50.33
SANTOSHVARAN	26	168	61	52.38	52.34	53.86	53.94	50.43	48.43	49.72	50.83	49.92	49.83	50.08	50.34

TO STUDY THE NORMATIVE DATAS OF VESTIBULAR EVOKED MYOGENIC POTENTIAL AND TO COMPARE WITH AGE AND SEX MATCHED MIGRAINE PATIENTS

Dissertation submitted to

The Tamil Nadu Dr. MGR Medical University

In partial fulfillment of the regulations

for the award of the degree of

M.D. PHYSIOLOGY

Branch V

INSTITUTE OF PHYSIOLOGY & EXPERIMENTAL MEDICINE

Government Madras Medical College and Hospital